G.P.-S.59968—1970-71—2 500

J 219

In the Supreme Court of South Africa
In die Hooggeregshof van Suid-Afrika

(APPELLATE. ~~Provincial~~ Division)
(~~Provinsiale~~ Afdeling)

Appeal in Civil Case
Appèl in Siviele Saak

P. BLYTH. *Appellant,*

versus

E. VAN DEN HEEVER. *Respondent*

Appellant's Attorney *Prokureur vir Appellant* WEBBER N. *Respondent's Attorney* *Prokureur vir Respondent* CLAUDE REID + H

Appellant's Advocate *Advokaat vir Appelland* ADV. BROWDE SC ADV. GALGUT *Respondent's Advocate* *Advokaat vir Respondent* ACKERMAN SC STAFFORD.

Set down for hearing on *Op die rol geplaas vir verhoor op* 11/5/79 – 17/8/79

(TPD)

CORAM: RUMPFF HR, CORBETT AR, JOUBERT AR, GALGUT AR, et. BOTHA AR

Adv. BROWDE: (1) 9h45 – 11h00 11h15 – 12h47, 14h15 – 15h~~45~~30
16h46 – 17h00
(2) 9h45 – 10h40
(3) 12h10 – 12h45

Adv. ACKERMAN: (1) 10h40 – 11h10; 11h25 – 12h45; 14h15 – 15h35
15h50 – 17h00
(2) 9h45 – 11h05; 11h25 – 12h10

UITSPRAAK HOF NR 1 27.9.79 @ 9.00 vm C A V

Bills taxed—Kosterekenings getakseer

Date Datum	Amount Bedrag	Initials Paraaf

Writ issued
Lasbrief uitgereik

Date and initials
Datum en paraaf

IN THE SUPREME COURT OF SOUTH AFRICA

(APPELLATE DIVISION)

In the appeal of:

PETER BLYTH ..... appellant

and

DR E. VAN DEN HEEVER ..... respondent

Coram: Rumpff CJ, Corbett, Joubert JJA, Galgut et Botha AJJA

Date of hearing: 15, 16 and 17 August 1979

Date of Judgment: 27 September 1979

J U D G M E N T

CORBETT JA:

At about 4.30 p.m. on Sunday 23 May 1971 the appellant (plaintiff below) sustained fractures of the bones of his right forearm (the radius and the ulna) as a result of a fall from his horse while playing polo. After receiving, at the polo field,

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first-aid in the form of the application of an L-splint to his arm and an injection, appellant was conveyed in a private motor vehicle to the provincial hospital at Ermelo, a distance of some 61 km. He arrived there at about 5.30 p.m. The family doctor at the time was the respondent (defendant below). He was called to the hospital and saw the appellant at about 6 p.m. Having examined the broken arm, respondent decided to perform a reduction of the fractures under general anaesthetic. Arrangements were made for an operating theatre to be made ready for this purpose at 8 p.m. that evening and for an anaesthetist to be available. At the appointed time the operation was performed. It ultimately took the form of an open reduction (i.e., a reduction involving a surgical incision in order to expose the fracture site) of both the radius and the ulna. In the case of the ulna the two bone fragments were aligned and fixed in position by means of a metal plate. Finally the arm was encased - from approximately the middle of

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the upper arm to the base of the fingers - in a plaster cast.

Appellant remained in the Ermelo hospital from then until the following Saturday (29 May 1971), when he was moved to the Rand Clinic in Johannesburg. By that stage a massive sepsis had destroyed most of the muscle tissue in the extensor and flexor compartments of appellant's right forearm and also certain of the forearm nerves. On the Sunday (30 May) a specialist orthopaedic surgeon, Dr Boonzaaier, who had treated appellant on Friday 28 May, told appellant's mother, Mrs M.E. Blyth (whom I shall call Mrs Blyth senior, in order to distinguish her from appellant's wife, whom I shall call Mrs Jennifer Blyth), that appellant "would be lucky if he retained 20% use of his arm".

This prognosis unfortunately proved to be unduly optimistic. Despite a week's treatment at the Rand Clinic, where appellant was attended by Dr Boonzaaier, the sepsis persisted. At the end of the week (i.e. on Saturday, 5 June) the appellant was allowed to go home. Thereafter he was seen

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once a fortnight by Dr Boonzaaier. There was, however, no material improvement in the condition of his arm. After he had seen two medical practitioners in Durban, appellant eventually consulted Prof. Louis Solomon, Professor of Orthopaedic Surgery and Chief Orthopaedic Surgeon at the University of Witwatersrand. This was on 28 August 1971. Prof. Solomon performed an operation on the arm on 2 September 1971 with a view to eliminating the infection. This was successful in that the infection cleared up after two or three weeks.

Thereafter, a colleague of Prof. Solomon's, a Dr Biddulph, who specialises in hand surgery, attempted certain reconstructive surgery aimed at restoring to some extent the nerve function in the forearm and hand. The operation was performed in two stages on 26 October 1971 and 25 January 1972. Prof. Solomon assisted at the first operation. These procedures produced very limited, if any, improvement in the condition of appellant's arm. Eventually the surgical wounds healed and the position became stabilized. At the time of the

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trial in the Court a quo the forearm had become reduced to what the trial Judge (ELOFF J) described as "a shrunken claw-like appendage of extremely limited functional value". ELOFF J elaborated upon this with a fuller description of the limb, reading as follows:

> "From the elbow downwards the forearm is bowed and extremely wasted from the proximal quarter down to the wrist. There is only one complete bone in the forearm, namely the radius; the middle portion of the ulna has been excised and the distal end of the proximal fragment has become united with the radius. The distal third of the ulna is intact but markedly osteoporotic. The wrist itself is fixed with 5 degrees of motion and the state is one of fibrous ankylosis in 15 degrees of flexion deformity. The knuckle joints are in a state of moderate hyperextension of the metacarpo-phalangeal joints in the second, third, fourth and fifth fingers. The thumb is in a state of absolute adduction with obliteration of the first intermetacarpal space by adduction of the thumb. All the proximal interphalangeal joints are flexed to about 80 degrees and fixed in that position except for a few degrees of passive flexion which is possible. The distal interphalangeal joints are in 30 degrees of flexion and also very largely fixed by fibrous tissue binding the tendons and the joints. Of the muscles a great deal has

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> been excised, and the only two with some functional value are flexor carpi radialis, and extensor carpi radialis. There is a complete loss of light touch and pinprick sensation over the volar aspect of the hand with patchy presence of poor quality sensation in the proximal areas of the hand. He has reasonable sensation of the forearm however. The features of the hand include median ulna nerve palsy with intrinsic paralysis."

On 17 May 1974 appellant instituted action in the Transvaal Provincial Division against respondent, claiming damages in the sum of R70 941 and costs of suit. Shortly before the trial, which commenced on 21 March 1977, this claim was increased to R112 123,56. After a lengthy trial ELOFF J granted absolution from the instance with costs. The present appeal is against the whole of the trial Judge's judgment and order.

Broadly speaking, appellant's case against respondent is that in treating him for the broken arm respondent acted negligently in that he failed to exercise the professional skill and diligence required of him, as a medical practitioner,

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in the particular circumstances of the case; that respondent's negligence in this regard caused or materially contributed to the functional disability affecting appellant's right arm and the pain and suffering which he had endured in regard thereto; and that respondent was consequently obliged, in delict, to compensate appellant in damages. At the trial a number of grounds of negligence, foreshadowed in the pleadings, were advanced in evidence. These grounds related to respondent's pre-operative treatment of the appellant, his decision to perform an open reduction of the fractures, his operative procedures, an alleged failure to keep proper records of the details of the operation and of appellant's condition while under his care, the type of plaster cast applied to the arm and his post-operative care of the appellant. Before this Court, however, appellant's counsel confined his case, on the negligence issue, to certain aspects of the post-operative care and treatment of the appellant. In so circumscribing the issues appellant's counsel, in my view, exercised a wise discretion. A reading

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of the evidence satisfies me that the other grounds were either not shown preponderantly to have constituted professional negligence or were not causally connected with the ultimate disaster which overtook appellant's right arm.

As I see it, this case resolves itself into three main questions: (i) what factually was the cause of the ultimate condition of appellant's arm; (ii) did negligence on the part of respondent cause or materially contribute to this condition in the sense that respondent by the exercise of reasonable professional care and skill could have prevented it from developing; and (iii) if liability on the part of respondent be established, what amount should be awarded to appellant by way of damages? I shall deal with these questions in the order in which I have posed them.

The cause of the ultimate condition of appellant's arm

Now, it is clear that had appellant not sustained a broken arm at polo he would not have ended with a useless

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"claw-like appendage". On the other hand, fractures of the nature sustained by appellant, if properly and timeously treated, do not usually have the dire results that these fractures did. They usually heal with little or no adverse after-effect. What went wrong in this case?

This was the question to which a major portion of the expert testimony was directed. And in order to appreciate the meaning and thrust of this evidence it is necessary to recount in some detail the known facts in regard to appellant's condition and the treatment given him from the time he entered the Ermelo hospital at about 5.30 p.m. on Sunday evening until he left for the Rand Clinic on Saturday morning.

When respondent saw appellant at the hospital at about 6 p.m. on Sunday, he removed the L-splint in order to examine the arm. The arm then telescoped in a concertina-like fashion and the radial pulse disappeared. This led respondent to conclude, probably quite rightly, that in an unreduced, unstabilized condition the fractures were likely to cause interference

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with the radial artery and with the proper flow of blood in that artery. He accordingly decided that it was necessary, as a matter of urgency, to reduce the fractures as soon as possible and in these circumstances the arrangements were made for him to operate at 8 p.m. Respondent asked the sister in charge, sister Greyling, to try to obtain X-ray photographs of appellant's arm before the operation. At that stage there was only one radiographer attached to the Ermelo hospital and it transpired that she could not be found that Sunday evening. Consequently when ~~respont~~ respondent came to operate at 8 p.m. there were no X-ray plates available. Respondent nevertheless decided to proceed. In this he was severely criticised by appellant's expert witnesses. He himself justified the decision on the ground that it was an emergency case and in this connection he was supported by the expert witnesses called on his behalf. He also performed the operation without the assistance of another medical practitioner (other than the anaesthetist). This was a

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further ground of criticism and a point of dispute between the experts. In view, however, of the aforementioned limitation of appellant's case in regard to negligence it is not necessary to resolve these issues.

Had pre-operative X-rays been taken it seems likely that they would have revealed a simple transverse fracture of the ulna in the region of the middle third and a comminuted fracture of the proximal third of the radius (i.e., the third closest to the elbow joint). At the trial respondent hotly disputed the contention that the radial fracture was a comminuted one, averring that it too was merely a simple transverse fracture. The Court nevertheless found that the radial fracture was a comminuted one, in the sense that the bone was broken in two places at the fracture site. Support for this conclusion was found in, inter alia, the evidence of Dr Spiro, a radiologist, who at the request of Prof. Solomon and on 28

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August 1971 examined the appellant and furnished a report on his findings. X-ray photographs taken at the time could not be traced, but the report, which was based on Dr Spiro's interpretation of the X-ray plates and from which he refreshed his memory when giving evidence, indicated two fractures of the radius, one situated 4,5 cms. and one 6 cms. from the proximal end of the radius. The rejection of respondent's evidence on this point raises a question of creditworthiness to which I shall refer later.

At the operation and once the appellant was suitably anaesthetized, respondent first attempted a closed reduction (i.e. a reduction by manipulation and without exposing the fracture site by surgical incision) of the ulna. After three unsuccessful attempts he decided that he would have to perform an open reduction. As a prelude to this he applied an Esmarch tourniquet to the upper arm in order to create a bloodless field in which to operate. Having expelled the blood from the arm, he made a surgical incision and exposed the frac-

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ture site. He aligned the bone fragments and plated them. He also exposed the radius by another surgical incision and aligned and engaged the fragments. Respondent's description in evidence of how he aligned two radial fragments contrasts with the description of the operation which he is alleged to have given to Mrs Blyth senior immediately after the operation. According to her, he said that the radius was "shattered" and that "he pushed them (the fragments) back into place to the best of his ability". She also stated that he appeared very worried. In view of the finding that the radius was in fact comminuted and that there were three fragments, Mrs Blyth's version appears to be the more probable one.

Both surgical wounds were sutured and the full-length plaster-cast, already described, was applied. Before putting on the plaster of Paris respondent wrapped the arm with cotton wool, which was to act as padding between the arm and the plaster. The operation lasted about an hour. Appellant was then returned to the ward.

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The only extant contemporary records of appellant's condition and treatment while in the Ermelo hospital are a series of so-called "bed-sheets" and a chart showing his temperature, pulse rate and respiration at certain regular intervals. The bed-sheets were kept in the duty room ("dienskamer") for the ward. Entries therein were made by the nurse who accompanied the doctor on his rounds or the nurse under whose care the patient fell. These entries purport to record the treatment given to the patient, including medicaments, the doctor's orders concerning the patient and the execution thereof, and any relevant observations concerning the condition of the patient. Several of the nurses who made entries in appellant's bed-sheets, viz. sister Le Roux, sister Greyling (now Mrs Du Toit) and sister Loots (now Mrs Van der Bergh), were called as witnesses. They confirmed their entries and in some instances sought to explain them. One of the staff who made entries, sister Froneman, died before the trial. Entries made by her were formally admitted by the parties as having been made.

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A similar admission was recorded in regard to an entry made by a sister Van Staden who was not called as a witness. As regards the entries made by sister Froneman, in general their content would seem to be admissible in evidence in terms of section 34(1) of the Civil Proceedings Evidence Act 25 of 1965. In so far as the statements comprising these entries might be said to contain expressions of opinion they would appear to be admissible in terms of the section provided that the expressions of opinion would have been admissible if made orally by sister Froneman in the witness box (cf. Dass v Masib, [1968] 2 All E.R. 226).

In addition to these records Mrs Blyth senior kept a diary recording her sons's accident, his treatment and his condition from time to time. She commenced this diary some two to four weeks after the accident. The portion covering the week while appellant was a patient in the Ermelo hospital was consequently not a contemporaneous record and could not be used to refresh Mrs Blyth's memory when giving evidence;

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but, as I shall show later, it is nevertheless of some evidential value. The later portion, which was kept contemporaneously was used by Mrs Blyth to refresh her memory.

These contemporary records are of some value because the witnesses themselves gave accounts of the clinical picture which were in some respects widely divergent. Some of these conflicts are not material to the ultimate issues in this case and need not be considered; others are and will have to be resolved. Nevertheless, subject to these disputes of fact, there emerges from the evidence of the various witnesses concerned, i.e. the nursing staff, the appellant, Mrs Blyth senior, Mrs Jennifer Blyth (who at the time of the accident was Miss Cook), respondent and his partner, Dr Van Niekerk, considered against the background of the contemporary records, the following general picture.

After being examined by respondent, and at about 6.10 p.m. on Sunday, appellant was given an injection of 100 mg. of Pethidine, an analgesic. At 7 p.m. appellant was given a

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further 100 mg. of Pethidine as part of his pre-operative medication. After his return to the ward, after the operation, appellant was examined by respondent. His arm was placed on a pillow and the forearm kept in a vertical position by means of a drip-stand. This was done pursuant to instructions to that effect given by respondent. The latter also told the nursing staff to administer Pethidine when required but at four-hourly intervals. At 10.20 p.m. appellant received another 100 mg. of Pethidine for pain. The bed-sheet records, at this point, that the patient was encouraged to move his hand and that the circulation was watched ("sirkulasie is dopgehou"). At 12 midnight appellant again complained of pain and was given two Doloxene tablets, an analgesic somewhat milder than Pethidine. At 2.30 a.m. on the Monday a further 100 mg. of Pethidine, together with 2 Doloxene tablets, was administered for what sister Loots described in the bed-sheet as "erge pyn". At the same time she recorded: "Vingers is baie styf. Kleur is goed. Kan nie duim roer nie. Slaap tussenin".

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At 7 a.m. on Monday appellant was taken to the X-ray department for X-ray photographs of his arm to be taken. After that he was examined by respondent. Respondent appeared unworried about the arm. Appellant complained that the plaster was too tight around his thumb and respondent cut away the plaster around the base of the thumb. According to appellant, respondent asked whether he could move his fingers, but he could not recall being able to do so, although they may have moved fractionally. Respondent himself did, however, straighten his fingers manually. Respondent, on the other hand, testified to appellant having had good motor function in his fingers. I shall refer to this and other disputes of fact in regard to the clinical picture during this week at a later stage.

Appellant stated that he suffered a lot of pain during Monday morning and that the pain got progressively worse during the day. He felt that the pain was caused by the plaster being too tight. During the day his fingers

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began to swell and by the afternoon "they were beginning to change colour, becoming a bluish colour". In this he is supported by his mother who was at his bedside for most of the Monday morning. She stated that appellant complained that his arm was very painful and that she noticed that by lunch-time his fingers were swollen and a "bit on the blue side". She told the nursing staff and asked them whether they did not think that the plaster was constricting his arm. The evidence about the fingers being bluish in colour was disputed by certain of respondent's witnesses (who included the nursing staff) and the trial Judge found that he could not find that this averment had been substantiated. This finding was not challenged on appeal.

On the other hand, there does not seem to be much doubt that by late Monday morning appellant's arm was swollen and painful. At 11 a.m. he was given 100 mg. of Pethidine for pain. At some time during the morning (this does not appear to have been recorded in the bed-sheet) sister Froneman

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telephoned respondent at his consulting rooms. It must be inferred that she did so because she was concerned about the condition of the appellant's arm, probably because of the pain and swelling. Respondent stated that he could not recall such a telephone call, but that the call was made appears clearly from the evidence of sister Greyling. The probability is that respondent did not react to this call. Thereafter sister Greyling herself telephoned respondent. She was worried that the swelling had not diminished and told respondent so. He instructed her to split the plaster and apply a crèpe bandage. This brings me to one of the major disputes of fact.

It was the evidence of appellant, Mrs Blyth senior and Mrs Jennifer Blyth that some time during Monday afternoon appellant's plaster was partially split by two slits about 3 inches in length being cut at the top of the plaster and two similar slits being made at the bottom in the vicinity of appellant's thumb and little finger. No crèpe bandage

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was applied. Appellant and Mrs Blyth senior stated further that on Tuesday afternoon (i.e., the following day) the plaster was split down its complete length. The plaster was opened slightly by means of a spreader, but the underlying cotton wool was not cut. A crèpe bandage was then applied. This version was denied by respondent. His version of the splitting, as formulated in his further particulars to his plea, as amended on 18 March 1977 (i.e., three days before the commencement of the trial), is that before 3 p.m. on the Monday his instructions were carried out by the plaster being split "down to the skin from the hand along the volar surface of the arm to the top of the plaster" and by the split plaster being kept in position by means of a crèpe bandage.

In support of his version respondent called sisters Le Roux and Greyling. During the course of her evidence-in-chief sister Le Roux was asked (with reference to the appellant):

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"Op watter ander geleentheid het u hom òf gesien òf behandel òf iets aan hom gedoen òf....?-- Die eerste keer wat ek hom gesien het was die 25ste, ek meen dit is Maandag die 25ste.

Ja?-- Toe het Suster Greyling my gevra om haar te help om die gips te verwyder.

Ek sien. En wat het u gedoen?-- Ek kan nie onthou of ek haar gehelp het deur die arm te ondersteun en of sy die arm ondersteun het dat ek die gips verwyder het nie, maar ek onthou wel dat ek haar gehelp het in die uitvoer van die split van die gips.

Nou u het gesê Maandag die 25ste. Nou Maandag was die 24ste?-- Ekskuus tog, dit is nou die dag.... jammer. Kan ek net weer kyk na die inskrywings.. ekskuus tog. Ja, die 24ste, jammer."

She thereafter proceeded to describe how the plaster was split completely with an electric saw, opened with a spreader and the underlying cotton wool cut to the skin with a special pair of scissors. This occurred between 1 p.m. and 3 p.m. Under cross-examination sister Le Roux was unable to explain why she spoke in chief of "Maandag die 25ste"; it was pure chance ("blote toeval"). She could not remember dates and in this regard had to rely on what

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appeared in the bed-sheet. She stated that she had an independent recollection of this particular splitting and mentioned that when the splitting had been done she noticed a red mark on the skin near appellant's elbow which was massaged and then seemed to disappear. She did not think that Mrs Blyth senior was present when the plaster was split.

At this point it is relevant to note that immediately after an entry recording the administration of 100 mg. of Pethidine at 11 a.m. on Monday the 24th, the following entry (for which no time is given) appears:

> "Hand nog geswel. Dr. E v.d. Heever in kennis gestel. Beveel - split gips, wend crepe verband aan. Bevele is uitgevoer."

It was signed by sister Greyling. The next ensuing entry is recorded as having been made at 4 p.m. so that it can be safely assumed that the entry quoted was made at some time between 11 a.m. and 4 p.m. on the Monday.

Sister Greyling (now Mrs Du Toit) stated in evidence-

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in-chief that, apart from the entries in the bed-sheets she herself did not have a very good recollection of the case, including the splitting of the plaster. This is plainly evident from the answer which she gave to respondent's counsel, when asked about the splitting:

> "As u 'n bevel kry 'split gips, wend crèpe verband aan', wat beteken so 'n bevel mevrou?--Ja, dit beteken om die gips heeltemal te split tot op die vel, maar ek is nie heeltemal..... ek kan my nie..... my geheue kan my nie..... ek is nie heeltemal seker maar dit is..... definitief het ek die gips... moes ek dit gesplit het want 'n crèpe verband beteken daardie gips was heeltemal gesplit van bo tot onder".

During cross-examination by appellant's counsel it transpired that sister Greyling had also been subpoenaed by appellant and had on 23 March 1977 (she gave evidence on 4 May 1977) given a statement in writing to appellant's attorneys. In this statement (the contents of which she admitted) she referred to a telephone call by sister Froneman on the Monday and then went on to say that she saw the appellant again on the Tuesday. His fingers were still swollen

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and because she was concerned she telephoned the doctor. Instructions were given to split the plaster and this was done. The statement included the following:

> "Dit is wel gesê in die verslag van Maandag dat die gips gesplit was, maar hoe weet ek regtig nie. Wat ek wel kan onthou is dat later in die dag en ook Dinsdag, dat die gips nog in posisie was. Daar is geen twyfel dat die gips heeltemal losgesny was op Dinsdag en nie die Maandag nie."

(The "verslag van Maandag" evidently referred to the relevant bed-sheet.) When all this was put to her she said that an error must have crept in: her dates were incorrect. It also transpired that after making this statement she attended a consultation with appellant's counsel at which a further statement was taken. This statement was read out to her and she admitted the contents. It is manifest from this statement as well that sister Greyling was at the time of the view that the plaster was fully split on the Tuesday. In this statement there is also a suggestion that the plaster might have been partially split on the Monday and fully split

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on the Tuesday. She also stated - and confirmed this in evidence - that if she had partially split the plaster her entry in the bed-sheet (referring apparently to the Monday entry) would have been precisely the same.

I have dealt with the evidence of the two nursing sisters on the question of splitting in some detail because, in my view, it demonstrates their unreliability on this issue and because there is discernible in the evidence of sister Greyling, at any rate, a measure of support for the appellant's version of the splitting.

Dr Van Niekerk, who was called to give evidence on behalf of respondent, stated that if there had been a partial splitting of the plaster, as described by appellant, he (Van Niekerk) would have noticed this when visiting appellant during the course of the week and that he had not noticed such a splitting. Dr Van Niekerk's evidence that he visited appellant on Monday and Tuesday was, however, rejected by the trial Judge (correctly, in my view) and consequently Dr Van Niekerk's evidence does not take this issue any further.

Respondent testified that when he gave instructions on Monday morning for the plaster to be split and a crèpe bandage to be applied, he expected the nursing staff to split the plaster completely down one side. A plaster may also be split completely both anteriorly and posteriorly. This procedure is known as "bivalving". Had he wished this latter procedure to be followed he would have told sister Greyling to "bivalve" (or "halveer") the plaster. A partial splitting, as deposed to by appellant's witnesses, would have served no purpose. He had never given an instruction for such a partial splitting to be executed; and certainly did not give it in appellant's case. On Tuesday morning when he visited appellant, the arm was in plaster with a crèpe bandage around it. He did not unwind the bandage but thought that his instructions had been carried out. He could not see whether the cotton wool padding had been cut to the skin, but on the Thursday when he removed the plaster he found that it had.

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This evidence contrasts starkly with versions of the splitting episode given in the further particulars to respondent's plea. I have already referred to the amended version which was introduced on 18 March 1977, shortly before the commencement of the trial, and which is in conformity with respondent's evidence. There had, however, been no less than three previous versions. In answer to a request by appellant that particulars be given of the treatment, attention and advice given by respondent to appellant during 23 to 27 May 1971 the following, inter alia, was stated by respondent's legal representatives on 6 August 1975:

> "Defendant examined Plaintiff on the 24th May 1971, at approximately 8 a.m. and cut away a piece of the plaster cast along the thumb.... At 11 p.m. on the same day Defendant instructed the nursing sister to split the plaster partially and put a crepe bandage around the arm..... On either the 25th or 26th May 1971 Defendant instructed the nursing sister to split the plaster cast fully". (My underlining.)

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I shall refer to this as the "first version". On 18 February 1977 (i.e. about a month before the commencement of the trial) notice was given on behalf of respondent that at the hearing, application would be made for the amendment of the further particulars by deleting the latter two sentences of the passage above and the substitution of the following:

> "At 11 p.m. on the same day Defendant telephonically instructed the nursing sister to split Plaintiff's plaster down to the skin from the hand along the volar surface of the arm to the top of the plaster and to keep the split plaster in position with a crepe bandage.
> .....
> On the 25th May 1971, at approximately 8 a.m. Defendant, on discovering that the cottonwool under the plaster had not been cut to the skin, although the plaster itself had been split as instructed by him, proceeded himself to cut the cottonwool to the skin."

I shall call this the "second version". And 5 days later, on 23 February 1977, notice was given withdrawing the notice of 18 February 1977 and indicating that at the hearing application would be made for the following to be substituted for the particulars in question:

> "At 11 a.m. on the same day Defendant telephonically instructed the Nursing Sister to split Plaintiff's plaster down to the skin from the hand along the volar surface of the arm to the top of the plaster and to keep the split plaster in position with a crepe bandage. Defendant's instructions were carried out, but the cottonwool under the plaster was not split.
>
> .....
>
> On the 25th of May, 1971, at approximately 8 a.m., alternatively, at approximately 3.30 p.m., Defendant proceeded himself to cut the cottonwool along its length down to the skin".

I shall call this the "third version". (In all these extracts "Plaintiff" refers naturally to appellant and "Defendant" to respondent.)

Apart from their mutual contradictions, these different versions, or the first three at any rate, conflict directly with the evidence given by respondent at the trial. For instance, the instruction to split partially on the Monday and fully on the Tuesday, referred to in the first version, is wholly inconsistent with respondent's evidence that he had never given an instruction for

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a partial splitting, that this would have served no purpose and that, as far as he knew, the plaster was split fully on the Monday. And the averment, in the second and third versions, that on Tuesday 25 May respondent himself cut the cotton wool to the skin after discovering that the nursing staff had failed to do this, cannot be reconciled with his evidence that on opening the plaster on Thursday he found that the nursing staff had cut the cotton wool as instructed.

Under cross-examination respondent was taxed with these conflicts and contradictions. He conceded that the various versions of the splitting contained in the further particulars and the amendments or proposed amendments thereto were based on information furnished by himself and he accepted responsibility therefor. He was nevertheless not able to furnish any acceptable explanation for the conflicts and contradictions. In fact some of the explanations which he attempted to give reflect adversely on his credibility. It should also be pointed out that respondent

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admitted that shortly after the action was instituted he fetched from the hospital all the records, including the bed-sheets, relating to appellant's case and that thereafter these remained in the possession of his attorneys. He, therefore, had access to these records when giving instructions for the compilation of the further particulars. Ultimately, under cross-examination respondent was forced to concede that parts of his recollection concerning the plaster were totally unreliable and that he would not dispute a partial splitting on Monday afternoon followed by a complete splitting on the Tuesday.

This is a convenient point at which to refer to another strange feature of the evidence adduced by respondent and some of his witnesses. In the course of his evidence-in-chief Dr Van Niekerk was asked about sister Froneman (deceased). He stated that she was a capable nurse and then his evidence proceeds:

> "Ja, en watse soort geaardheid het sy gehad, hoe sou u haar beskryf? In haar verplegingshoedanigheid?-- Ek dink sy was / miskien.....

> miskien 'n bietjie geneig tot .... om 'n bietjie melodramaties te wees, sy het dinge 'n bietjie ernstiger gesien as wat dit miskien sou wees. Gou op hol te raak as ek dit so mag uitstel (sic)".

Similar observations were elicited from sisters Le Roux and Greyling and from respondent himself. Sister Le Roux said that sister Froneman was inclined to exaggerate and call for help, sometimes unnecessarily. Sister Greyling said that she was over-cautious and would telephone the doctor unnecessarily. Respondent stated that she was slightly inclined to be melodramatic, over-cautious. When respondent was asked why this evidence had been led and whether it was not an attempt, in anticipation, to water down the significance of sister Froneman's telephone call on the Monday morning, he responded angrily (so it is to be inferred): "Dit is 'n infame leuen". Incidentally, it is to be noted that under cross-examination sister Greyling stated that had sister Froneman not telephoned when she did she (sister Greyling) herself would have done so.

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The trial Judge gave full attention to the issues concerning the splitting of the plaster and pointed out that they involved questions of credibility. He recorded his impressions of some of the witnesses involved. As to Mrs Blyth senior he stated that a great deal of time and attention had been devoted in cross-examination and argument to discrediting Mrs Blyth. He concluded, however —

> "She appeared to me to be a lady of character and refinement and I never had the impression that she endeavoured to be anything other than honest in this case. I reject any suggestion of an endeavour to mislead. That, however, does not mean that she might not be genuinely mistaken in her impressions ..... ".

As regards appellant and Mrs Jennifer Blyth he stated —

> "The plaintiff also and certainly Jennifer seemed to be honest witnesses. It is true that plaintiff exaggerated, badly at times, for example in saying that on Wednesday his fingers looked like rotten bananas, and it says much for Mrs Blyth's honesty that she was unwilling to support him therein.

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> That tends to make me a little wary of, in my view, any unsupported evidence that he gives regarding the nature and extent of his symptoms. Nevertheless I do not think that he or she ever consciously fabricated."

(Appellant's evidence as to his fingers looking like "rotten bananas" actually referred to the Tuesday morning.) The learned Judge did not record his impression of sisters Le Roux and Greyling, but he dealt fully with respondent's evidence on this issue and the protean character of his further particulars. He held that respondent was —

> " very confused and totally unreliable as to precisely what happened, save that his averments left an undertone of something having gone wrong in relation to his instructions."

The learned Judge's finding on this issue was as follows:

> " Summarising then I find that on the probabilities there was more than usual swelling to put it no higher in the forearm and fingers on the Monday and the Tuesday and the cast was split on the Monday afternoon but not the cotton-wool, and the cottonwool was tight and closely enveloped the arm."

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He thus rejected the evidence of a partial splitting on the Monday and a full splitting on the Tuesday.

With respect, I am unable to agree with this conclusion. It seems to me that the probabilities point overwhelmingly to there having been a partial splitting on Monday, followed by a complete splitting on the Tuesday. I say so for the following reasons:

(1) It is difficult to postulate that appellant, Mrs Blyth senior and Mrs Jennifer Blyth were mistaken on this issue. Their version involved two distinct cutting operations. Appellant, though still heavily sedated on the Monday, was aware of both operations taking place. Mrs Jennifer Blyth witnessed the partial splitting. Mrs Blyth senior saw on the Tuesday morning that a partial splitting had taken place and witnessed the full splitting later that day. If, as the learned trial Judge found, they were not persons

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who would consciously fabricate, then the probability is that their evidence on this point is correct.

(2) Then there is the extraordinary coincidence that in his first version (in his particulars) respondent gave virtually the same account of a partial and full splitting of the plaster as was given by appellant and his witnesses in evidence. Now, when respondent furnished the information upon which the first version was formulated he had no knowledge of what the appellant's case on this issue was going to be. This he admitted. On the other hand, it is clear that appellant's version, certainly as voiced by Mrs Blyth senior, was not tailored to fit in with respondent's first version because it appears that appellant's version was recorded in Mrs Blyth's diary at least four years before respondent's first version was put out. It is at least clear that a partial

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splitting followed by a full splitting was an unusual - and possibly in the circumstances an incorrect - procedure; and consequently the chances of both parties independently giving this version would be remote in the extreme, were it not true. I have little doubt that it was true. These considerations are not referred to by the learned trial Judge.

(3) As I have indicated, the evidence of the nursing sisters on this issue was unreliable and that of respondent totally so (as was found by the trial Judge). There is in fact in portions of sister Greyling's evidence some support for the appellant's version. Consequently there is little or no reliable evidence to gainsay that of appellant and his witnesses.

(4) In coming to the conclusion to which he did, the trial Judge was influenced by two factors: (a) the entry in the bed-sheet (quoted above) concerning

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the splitting and (b) the apparent pointlessness of such a splitting. I do not think that the entry in the bed-sheet is of sufficient significance to outweigh the probabilities to which I have already alluded. In fact this point is virtually neutralised by the admission of sister Greyling, who made the entry, to the effect that if there had been a mere partial splitting she would have made the same entry. As regards the pointlessness, it may be accepted that if the object of the splitting was to avoid vascular and circulatory problems (to which fuller reference will be made later), then a partial splitting was pointless; but if the object was merely to relieve discomfort then it might have served some purpose. In any event, there are all kinds of possible reasons why only a partial splitting was done and an enquiry into the purpose thereof takes one into the realms of speculation.

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(5) It is true that there is no entry in the bed-sheet recording a complete splitting of the plaster on the Tuesday. If, however, this ought to have been done on the Monday and the position was merely remedied on the Tuesday, this might explain the absence of such an entry.

(6) It has been suggested that because a crèpe bandage was applied on Monday, there must have been a complete splitting then. There is a dispute as to when the crèpe bandage was first applied, but even assuming that it was applied on the Monday I do not think that it necessarily follows that there must have been a complete splitting on Monday. Indeed in respondent's first version he stated that the initial instruction (on Monday) was to split the plaster partially and apply a crèpe bandage.

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For these reasons I conclude that whatever respondent's instructions may have been, the plaster was only partially split on the Monday and fully split on the Tuesday. As regards the cutting of the cottonwool, I am of the view that, as stated by appellant, this was not cut on the Tuesday (this appears to be the conclusion of the trial Judge too) and that it was only split when the plaster was eventually removed on Thursday morning.

After this lengthy digression I return now to the history of appellant's treatment in the Ermelo hospital. The bed-sheet for Monday the 24th records that at 4 p.m. appellant complained of pain in his hand and arm and was given two Doloxene tablets. This was repeated at 9 p.m. that evening. Sister Loots recorded, apparently during the night, "Slaap redelik goed - tussen vingeroefeninge". At 8 a.m. the following (Tuesday) morning appellant was visited by respondent. According to appellant his fingers were at that stage very swollen and he was suffering a lot

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of pain. He told respondent that he was having discomfort caused by the tight plaster. Respondent asked him to move his fingers but he could not recall being able to do so. Respondent himself tried to move them. Respondent was generally reassuring and said that appellant would be able to go home on the Thursday. According to respondent the appellant looked very much better on Tuesday morning. The swelling of his fingers was definitely slightly less, the colour was good and the routine tests which he carried out (about which more anon) were favourable. Again very conflicting evidence.

According to the bed-sheet respondent gave no new instructions on that visit. At 2 p.m. appellant was given 2 more Doloxene tablets for pain. At 2.30 p.m. it was recorded: "Kla van erge pyn en ongemak - verband verpak, watterol baie styf om arm." At the same time appellant was given two more Doloxene tablets. Mrs Blyth senior visited appellant on Tuesday. She arrived at about

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9 a.m. and stayed with him until about lunch-time. She noticed that he was in great pain and that his fingers were becoming more swollen and puffy. She became greatly concerned about appellant's condition and after lunch she went to see respondent at his consulting rooms. She explained to him that appellant was in great pain and that she thought that the plaster was very tight. Respondent appeared surprised to hear that the plaster had not been split all the way down and undertook to telephone the hospital immediately and instruct the nursing staff to do this. Mrs Blyth senior also asked him whether he (respondent) did not think that appellant should be taken immediately to see Prof. Solomon for specialised treatment, but respondent replied that this would not be necessary because appellant would be well enough to go home on Thursday. (It appears that Mrs Blyth knew of Prof. Solomon because he had already operated successfully on a member of her family.) Respondent agreed that this visit had taken

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place but gave a different account of the conversation. He stated that she told him that the appellant was uncomfortable and feverish. He could not recall the calling-in of a specialist being discussed. He denied her evidence about the splitting of the plaster.

Respondent nevertheless went to the hospital and visited appellant. This was at about 3.30 p.m. He was satisfied with appellant's condition and issued no fresh instructions. In view of the fact that the appellant's temperature chart (as interpreted in the light of sister Greyling's evidence) would have shown a reading of 102°F at 2 p.m. that day, respondent's attitude seems a little surprising; but nothing has been made of this.

Appellant's temperature remained relatively high during Tuesday. At 8 p.m. he was given two Disprin tablets and the sister in charge, sister Heyns, telephoned respondent's home. He was not there at the time, but received a message later and came to the hospital at about 10 p.m.

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He examined the appellant and diagnosed sepsis in his arm. He prescribed antibiotic medicaments, to be administered six-hourly.

During the night of Tuesday/Wednesday the appellant was feverish. His temperature appears to have remained at about 102°F and he began to perspire a lot. It is noted in the bed-sheet, against the time 6 a.m. on Wednesday, that his operation wounds were suppurating and had drained through the bandage covering his arm.

The remainder of the week may be described quite briefly. The sepsis which had become apparent on Tuesday persisted and in fact, as I have indicated, only cleared up completely some 4 months later towards the end of September. Appellant was visited by Dr Van Niekerk on Wednesday morning and by respondent in the evening. In the afternoon Mrs Blyth senior, who had been with her son during the morning, again went to see respondent in his consulting rooms. She was very concerned about her son's condition and again raised

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the question of a specialist, but respondent said that it was not necessary. Respondent does not recall this visit. At 8 a.m. on Thursday respondent decided to examine the wounds. He removed the plaster and the underlying cotton wool. He found that the ulna wound was suppurating. He removed alternate stitches and took a swab for analysis purposes. He cleaned the wounds, applied dressings and then put on a new plaster with a window over the wound areas so that the wounds could be watched and dressed.

Respondent had arranged to go away for the long week-end and handed appellant over to the care of his partner, Dr Van Niekerk, during his absence. He actually left Ermelo sometime during Friday. Respondent had also arranged for appellant to be seen and treated by Dr Boonzaaier, who visited the Ermelo hospital once a fortnight, usually on a Wednesday. This week, for some unexplained reason, he came on the Friday. At about 11 a.m. on Friday appellant was taken to the theatre and there, under general

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anaesthetic, Dr Boonzaaier removed the plaster, opened up the wounds and cleaned them. In this operation he was assisted by Dr Van Niekerk. Respondent was present and watched the operation. Dr Boonzaaier did not give evidence - a matter upon which I shall enlarge later - and consequently for a description of the operation and what was found the Court is dependent upon the evidence of Dr Van Niekerk and respondent. From their evidence it appears that in order to clean the wounds Dr Boonzaaier had to drain and remove a large quantity of pus and dead tissue, the appearance of which was described by Dr Van Niekerk as being "nekrotiserende kaasagtige materiaal". The infection was evidently very wide-spread and had invaded both the anterior (or flexor) and the posterior (or extensor) compartments of the forearm. So extensive was the infection that a mass of pus literally poured out of the forearm when the wounds were opened up, while the remaining pus and dead tissue was scraped out by Dr Boonzaaier, using his finger.

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This process exposed the ulna. The wounds were then cleaned and dressed and a new plaster applied. Next day appellant was removed to the Rand Clinic.

By the time that Prof. Solomon examined the appellant for the first time (i.e. on 28 August 1971), the appellant had no muscle action in his hand and there was no sensation in the skin. During the operation performed by him on 2 September 1971 he removed septic and devitalised tissue from the forearm and about 4 inches of dead bone from the ulna. At the operation performed by Dr Biddulph on 26 October 1971 it appeared that about 6 inches of the median nerve below the elbow and over the front of the forearm was totally missing. It seems probable that this sloughed off during ~~the~~ a surgical toilette performed by Dr Boonzaaier, either the one on Friday 28 May 1971 or one performed on a subsequent occasion. Moreover, the likelihood is that the bulk of this muscle and nerve damage had occurred by Friday 28 May, although, because of the sepsis, it was an

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on-going process and further damage would have occurred in the ensuing months before the sepsis finally cleared up.

Having thus traced the history of appellant's stay in the Ermelo hospital, it is now possible to consider the rival medical theories as to what happened to his arm. The expert evidence on this portion of the case is very voluminous and generally I am not able to do more than indicate my conclusions on the various issues raised thereby.

It is common cause that appellant's forearm was invaded by a massive sepsis. The general consensus appears to be that the micro-organisms which brought about the sepsis were probably introduced into the arm at the time of the operation on Sunday night and by reason of the surgical incisions then made. It is no part of appellant's case that in so introducing the sources of the infection, or in failing to prevent their introduction, the respondent acted negligently. The sepsis must, therefore, be regarded

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as a causal factor which is factually relevant but legally neutral. Appellant's case, however, broadly speaking, is that it was not sepsis alone, but sepsis operating upon and in conjunction with a very serious ischemic condition in appellant's forearm that caused the eventual catastrophe. In outline, the theory is that this ischemic condition developed shortly after the operation, that it gained in intensity during Monday and Tuesday and that by about 6 p.m. on Tuesday irreversible damage on a large scale had been caused to muscle and nerve tissue in appellant's forearm. This dead, or necrosed, tissue, together with damaged tissue at or near the fracture sites, was particularly vulnerable to the invading micro-organisms and formed a ready medium for the rapid and extensive spread of the infection. Respondent's case, on the other hand, is, broadly, that there was no large-scale ischemia, but that sepsis alone or sepsis operating initially upon the limited tissue necrosis at or near the fracture sites (the so-called

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"limited tissue necrosis" theory) were the sole causes of the ultimate condition of appellant's arm. It is to this basic issue that I now turn.

Ischemia, sepsis and the limited tissue necrosis theory

"Ischemia" means a deficiency of blood in a particular part of the body due to a constriction or occlusion of the blood vessels supplying that part. The most important function of blood is to supply oxygen to the tissues. Tissues cannot survive without oxygen. Consequently a protracted ischemia can cause the death of tissue.

Turning specifically to the muscles and nerves of the forearm there are basically two ways in which an ischemic condition of these parts can develop. The one is where an artery or major blood vessel serving the forearm becomes injured or constricted or occluded. The other is where a condition, referred to in evidence as a "compartmental syndrome", develops.

/ Anatomically...

Anatomically the forearm contains principally two bones, the radius and the ulna, which run roughly parallel and longitudinally down the forearm; a complex of muscles; and various nerves, arteries and other blood vessels. Each muscle is enveloped by a fairly loose sheath of connected fibrous tissue known as a "fascia". Groups of muscles are further enclosed in more dense and slightly elastic sheaths. The whole forearm is enclosed by the "deep fascia", a continuous sleeve of dense, inelastic connected tissue, situated just below the skin and the underlying subcutaneous tissue. "Septa", or dividing walls of fibrous tissue, joining the deep fascia to the ulna and the radius, and the interosseous septum, which forms a link between the ulna and the radius, cause the forearm to be divided into two main osteofascial compartments. These are known as the anterior (or volar or flexor) compartment and the posterior (or dorsal or extensor) compartment. The nerves and arteries serving the forearm and the hand are all contained in these different fascial compartments. The

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main artery of the upper arm, the brachial artery, divides just below the head of the radius into two main branches, the ulnar artery and the radial artery. Just below this division the radial artery gives off a leash of muscular branches and an interosseal artery, which in turn subdivides. The three main nerves of the arm are the ulnar nerve, the median nerve and the radial nerve.

As stated earlier, one of the ways in which an ischemic condition can develop in the forearm is a process known as a compartmental syndrome. The bodily mechanisms which cause this condition are not completely understood, but it would seem that it is always associated with a pressure build-up in the fascial compartments. There are a number of ways in which this process may be triggered off, but I shall confine myself to that which is relevant in the present case viz. a traumatic injury to the forearm resulting in bone fractures and muscle tissue damage, followed by manipulative and surgical damage to muscle tissue. The

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damaged tissues develop an inflammatory response and a fluid exudate begins to accumulate within the injured muscle and in the interstitial spaces. This accumulation of fluid increases the hydrostatic pressure of the interstitial fluid and this impedes the drainage from the venous end of the capillaries. Capillary pressure, at its venous end, therefore rises and a stage is reached when there is little or no return of fluid to the capillaries, resulting in an increase in interstitial fluid. This increase of interstitial fluid will become critical where the tissues cannot swell, as in the case of muscles contained in an unyielding osteofascial compartment. In such a situation the interstitial pressure eventually approaches the hydrostatic pressure within the capillaries. The transmural pressure (or pressure differential) across the capillary wall falls and the wall itself becomes unstable. If the transmural pressure falls below a certain critical level the vessel itself will close. Such closure occurs

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first with the smallest vessels. But a vicious circle is set up. As the ischemia caused by the closure of the smallest vessels increases, the permeability of the capillary wall increases and more fluid will escape from the vascular septum and accumulate in the interstitial compartments. This increases the interstitial fluid pressure and more and more blood vessels become occluded. The ischemic condition advances and, if unchecked, may result in the death of muscle tissue in the forearm on a large scale. At the same time the nerves of the forearm which are closely associated with the affected muscles may also be injured or even become necrosed. Eventually a stage may be reached when virtually the whole forearm has been necrosed by ischemia. The muscles become inert and are eventually replaced by hard, fibrosed tissue. During the process the affected muscles contract and the eventual result is that the fingers of the hand which are controlled by those muscles become fixed in a claw-like,

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flexed position, known as a contracture. For some reason that is not altogether clear, the anterior osteofascial compartment, which contains the flexor group of muscles, is more vulnerable to such an ischemic condition than the posterior compartment and this would appear to be the reason why in the ultimate contracture the fingers assume a flexed position. In such a contracture there is also a limited movement of the wrist. The same end result may be brought about in a similar manner, where the ischemic condition develops in the other manner previously mentioned, viz. by the injury to or constriction or occlusion of an artery or major blood vessel.

This type of condition was first described in 1872 by a certain Richard von Volkmann and for this reason it is generally referred to as a Volkmann's ischemic contracture. Since Von Volkmann's day medical learning on the subject has increased considerably. Much has been written about it, particularly in recent years, and medical students are

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taught of the dangers of a Volkmann's ischemic contracture (henceforth for the sake of brevity I shall refer merely to a "Volkmann's") developing. It is nevertheless a relatively rare occurrence and, in fact, various eminent medical experts who gave evidence in this case appeared to have had either limited or no direct experience of the condition.

The differentiation between a Volkmann's arising from injury to or constriction or occlusion of an artery or major blood vessel and that arising from a compartmental syndrome was fully discussed by one Holden in an article published in 1975. He labelled the former type 1 and the latter type 2. In the evidence the term "Holden type 2" was used apparently as an equivalent of the compartmental syndrome.

It appears from the literature on the subject that a Volkmann's resulting from traumatic injury (such as a bone fracture) is most likely to develop in the lower leg or in the upper arm or upper forearm. Consequently

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medical practitioners treating, inter alia, fractures of of the upper forearm must be on their guard against the possible development of an ischemic condition leading to a Volkmann's. They must watch out for the signs and symptoms of an impending ischemia and, if these signs present themselves, take remedial action. The classical symptoms are summed up in what have been described as the "five P's": pain, pallor, pulselessness, paralysis and para-anaesthesia (loss of sensation over and below the ischemic area). Depending on the type of ischemia involved, these symptoms may vary in their incidence and intensity. Thus, for example, the symptom of pulselessness may not present itself, initially at any rate, in the case of a Holden type 2; and there are recorded instances of a Volkmann's having developed without the pain symptom or in a relatively painless manner.

Once the threat of a Volkmann's has been diagnosed or is suspected, remedial action must be taken. Since the

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ischemic condition in the affected limb is, in the case of a Holden type 2 or compartmental syndrome, the result of a pressure build-up in the forearm, the most important remedial action in this case is to try to achieve a decompression. If the limb is encased in a circumferential plaster cast, then this must be split and, if necessary, removed. There was considerable debate between the experts as to the real extent to which a plaster cast may contribute to a compartmental syndrome, particularly where there is a padding of cotton wool between the plaster and the limb; but, whatever the decompressive effect of the removal may be, it is necessary that this should be done; firstly, in order to make a proper diagnosis and, secondly, as a prelude to more drastic action, if that should prove necessary. If the removal of the plaster, gentle massage and other treatment does not bring the necessary relief, then an operation known as a "fasciotomy" must be performed. This involves, in the case of the forearm, a surgical splitting of the deep fascia down the length of the forearm in

order to remove the compressive effect of this inelastic sleeve upon the tissue, blood vessels and interstitial fluids contained in the osteofascial compartments.

The evidence indicates that the build-up of an ischemic condition of this nature (i.e. the compartmental syndrome) may be very rapid or it may be a slow, insidious process. It starts with the tiny blood-vessels at the extremities of the vascular system (what one of the experts termed the "vascular tree") and as more and more blood-vessels become occluded, it works its way towards the larger blood-vessels and eventually spreads throughout the fascial compartment. As I have indicated, it is appellant's case, and the view of his experts, that the onset of the alleged ischemia in this instance was a fairly rapid one and that by 6 p.m. on Tuesday 25 May it had done its damage.

I turn now to the question of sepsis. Analysis of the swab which respondent took on Thursday 27 May, when

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he removed the plaster and cleaned the wounds, revealed the presence of a micro-organism known as staphylococcus aureus. No other infective organism was found in the specimen. Although the expert evidence was not altogether harmonious on most of the matters relating to sepsis, it was generally agreed that normally the staphylococcus aureus does not produce an invasive infection or cellulitis. It usually becomes localised by the body's natural defensive responses to infection and the sepsis takes the form of an abseess, such as a boil or carbuncle or a skin infection. It is also generally found in the subcutaneous tissue and not in the deep-seated tissue. It may, however, become invasive if it encounters tissue devitalised by, say, an ischemic condition. Its behaviour was described by Prof. Solomon as follows:

> " .... we must be quite clear about the normal behaviour of the staphylococcus. It will spread in the dead tissue, but if as you... the phrase you have used is limited tissue necrosis. If there
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> / is.....

> is a limited area of muscle damage due to the fracture, that is where it will spread, that is where it will make its abscess. If the muscle beyond that is healthy and is not suffering from the effects of ischemia, it will stop there, it will become enclosed and it will make an abscess, it will make a boil deep inside the arm, that is the classic behaviour of the staphylococcus. If, on the other hand, ischemia has preceded the situation and there was extensive muscle death beyond that point, then I would agree with you that the staphylococcus could spread in a wild way."

The staphylococcus aureus does not normally attack healthy muscle tissue, let alone healthy nerve tissue. Nerve tissue, of all the structures in the arm, is the least sensitive to infection. In fact Prof. Solomon stated that —

> "We see as orthopaedic surgeons in the course of a lifetime of practice, literally thousands of infections in the limb and I cannot recall seeing a staphylococcal infection produce death of the nerve. The nerve escapes, you just don't see it.

(In passing I might just say that of the various experts who gave evidence at the trial Prof. Solomon appears to

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have made a considerable impression on the trial Judge, who stated:

> "To the views of Professor Solomon I attach considerable importance, not only because of my impression of his astuteness, learning and balance but also because, of all the witnesses , excluding defendant and Van Niekerk, he was the first on the scene in that he saw the plaintiff in August 1971 when the wound over the ulna was still open.")

Although the aforegoing represents the normal characteristics of the staphylococcus aureus, virulent, invasive strains of this organism are occasionally encountered, especially in hospitals. They spread by invading the tissue planes between the muscles (they do not cause the death of the muscle cells), enter the bloodstream and cause septicaemia and pyaemia (a generalised form of septicaemia). Toxaemia ensues and the patient becomes very ill. He suffers shock and his pulse rate increases.

There are other types of virulent infection which can cause the rapid and invasive destruction of tissue.

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One is cloustridial myonecrosis or gas gangrene caused by an anaerobic organism. It is an overwhelming and frequently fatal disease, unless treated timeously by the surgical excision of the necrotic tissue or the amputation of the affected limb. It attacks muscle. Another is acute streptococcal gangrene (commonly called Meleney's gangrene). This is the product of a combination of organisms, acting synergistically (i.e. in a joint manner so that their combined effect is greater than the sum of their individual effects). This infection spreads in the subcutaneous tissue and produces a skin gangrene. It frequently bares the muscles without involving them. Mention was also made in evidence of an anaerobic streptococcal infection which apparently can produce a result similar to gas gangrene. It causes a type of toxaemia, shock and symptoms in the patient which are profound and easily detectable.

Finally, the so-called "limited tissue necrosis" theory which was advanced by respondent's experts as a

possible explanation for the invasive infection of appellant's arm by organisms of the staphylococcus aureus variety was succintly stated by ELOFF J as follows:

> "The theory as I understand it was this. In every fracture a certain amount of local lesion to surrounding tissue occurs with resultant haemorrhage and haematoma. This establishes a suitable culture medium for infective organisms which might be introduced by any effort at open reduction. The consequent inflammatory infective process results in exudation of fluid from the vascular bed into the interstitial tissue planes; the cellulitis may result in increased tissue pressure which is responsible for the slowing down of blood flow in the traumatised area, that in turn may be conducive to intravascular thrombosis in the venules and capillaries which may lead to cellular anoxia and necrosis. The process may then become an ongoing one until the body mechanisms can overcome the infection. The manifestation of the process is - so I understand the evidence - such that even if the organism is not of the virulent spreading staphylococcus aureus strain, a condition indistinguishable from the Holden type (ii) syndrome can be revealed."

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It now remains to resolve these conflicting views and decide what caused the ultimate condition of appellant's arm.

THE CAUSE

In determining what in fact caused the virtual destruction of appellant's arm, the Court must make its finding upon a preponderance of probability. Certainty of diagnosis is not necessary. If it were, then, in a field so uncertain and controversial as the one which I have thus far endeavoured to delineate, a definitive finding would become an impossibility. Bearing in mind that in this case appellant bears the burden of proof, the question is whether it is more probable than not that large-scale ischemia, coupled with sepsis, caused the damage (cf. Ocean Accident and Guarantee Corp. Ltd. v Koch, 1963 (4) SA 147 (AD), 157).

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I shall deal first with the theory, advanced by respondent's experts, that sepsis, and sepsis alone, could have done the damage. It seems to me that there are very substantial probabilities against this theory. The initial question, of course, is to decide what the nature of the infection is likely to have been. Gas gangrene and the streptococcal gangrene may be ruled out immediately. This was virtually common cause between the experts. The symptoms of these types of infection were not present. As regards the anaerobic streptococcal infection, Prof. Solomon was of the opinion that the type of toxaemia and shock produced thereby did not fit in with appellant's clinical picture. There seems to be substance in this viewpoint. Respondent's experts, particularly Dr Wienand and Dr Van Wyk, advanced the theory of a polymicrobic infection. Dr Wienand conceded, however, that the mixed organism produced a foul-smelling sepsis, whereas a staphylococcal infection is odourless. There is no evi-

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dence whatever that the pus removed from appellant's arm by Dr Boonzaaier on Friday 28 May possessed this foul-smelling characteristic. Neither Dr Van Niekerk nor respondent, who were present at this operation, suggests this. Dr Boonzaaier himself, though a signatory of the joint report submitted by respondent's experts and though present in Court for much of the hearing, was not called by respondent. Had there been such an odour, supporting the theory of a polymicrobic infection, I have no doubt that Dr Boonzaaier would have been called to depose to this fact. I think it may be taken as a fact that the sepsis was an odourless one, which points to the staphylococcus aureus. Moreover, staphylococcus aureus was the organism actually found on the swab. This establishes that this organism <u>was</u> present in appellant's arm. It may not establish conclusively that it was the only infective organism in the arm. Other organisms which may have been present may not have been taken up on the swab or may not have survived the journey

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to the analyst's laboratory. Account must also be taken of the effect of the anti-biotic medicaments administered. Nevertheless, taking all in all, the probabilities show, in my opinion, that the organism responsible for the sepsis was staphylococcus aureus. This appears to be in accordance with the conclusion of the trial Judge who held that, while the possibility of infection from sources other than staphylococcus aureus could not be excluded, such other organisms as "joined in" were not the gangrenous or streptococcal variety, were for the most part effectively dealt with by the anti-biotic medicament and were not more virulent than the staphylococcus aureus.

Staphylococcus aureus having thus been identified as the infective organism, the question is whether, unaided by an ischemic condition causing extensive tissue necrosis, it could have caused the damage to appellant's arm which was evident on Friday 28 May. It is clear

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from what I have said earlier in regard to the characteristics of this organism that such invasive tissue destruction is normally quite alien to its nature. The normal result of a staphylococcal infection is localised sepsis, generally contained within the subcutaneous tissue. There is, of course, the virulent strain, referred to by the Judge a quo as a "rara avis". The mere fact that it is of rare incidence inevitably tends to cause a mounting of the probabilities against it having been the culprit; but, apart from this, the characteristic behaviour of this rare strain, viz. invasive spreading in the tissue planes, generalised septicaemia and toxaemia, and the symptoms of extreme illness which it produces, do not fit into the clinical picture in this case. If sepsis alone were to have caused, in such a short space of time, the amount of damage that was evident on the Friday, it would according to Prof. Solomon, have had to have been one of an unrelenting, overwhelming kind. The witness stated "unreservedly" that the appellant's pulse chart

was not the sort of chart to be expected with an infection of this sort. Prof. Du Toit was of the same view. With reference to appellant's pulse chart he stated —

> "This is not the typical pyrexial and toxic pulse rate of a serious infection".

Attempts were made by certain of respondent's experts to read into appellant's temperature and pulse charts evidence of marked variations, corroborative of such extreme illness, but, in my view, they are not persuasive.

Another factor of cardinal importance in weighing the question as to whether sepsis alone could account for the damage is the fact that there was what the experts have described as a "selective" destruction of muscle and nerve tissue. Some muscles were left partially intact, the remainder destroyed; there was also selective damage to the main nerves. Dr Cooke, a vascular surgeon called on behalf of appellant, explained how this selective destruction fitted in with the concept of a Volkmann's with

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superimposed sepsis and expressed the view that it was most unlikely that infection alone would selectively damage muscle and nerve tissue in this way. I shall elaborate later on the extent to which the selectivity of the damage supports the theory of appellant's experts, but for the moment it is relevant to note that respondent's experts did not appear to furnish any real answer to this point.

The final point in this connection is the resistance of healthy nerve tissue to infection. I have already referred to Prof. Solomon's evidence in this regard. Dr Cooke's evidence was to the same effect. Respondent's experts, while apparently less ready to acknowledge the power of resistance of healthy nerve tissue to infection, seemed to concede that the wholesale destruction of nerve tissue in a forearm by infection, unaided by an ischaemic condition, would at least be an unusual occurrence.

The trial Judge, after a similar review of the

/ evidence....

evidence and having ruled out other types of infection, came to the conclusion that —

> " .... the probabilities are fairly heavily against staphylococcal infection destroying most of plaintiff's arm were it not for the fact of significant Ischemia having been caused."

I agree with this conclusion, save that I would assess the probabilities against the "sepsis only" theory as being even more weighty than ELOFF J appears to have regarded them.

As regards the alternative cause postulated by respondent's experts, viz. the limited tissue necrosis theory, I am in general agreement with the conclusion reached by ELOFF J, which was as follows:

> "I think that I can with reasonable confidence go along with the views of the plaintiff's experts that in a case such as the present the traumatic tissue death would be limited in extent; while tissue lesion and necrosis as I previously pointed out could well have a contributory effect in inducing swelling and may thereby lead to Ischemia, it is unlikely to engender

/ spreading....

> spreading infection except if the Ischemia renders theretofore undamaged tissue devitalized or defenceless; or except if that rara avis, the virulent spreading strain of staphylococcus aureus is the infective organism. So, except in that rare eventuality, one is back to Ischemia being a necessary component in a process - and that is what this case is all about".

So much for sepsis and the limited necrosis theory; and now I turn to appellant's theory of sepsis superimposed upon a Volkmann's ischemic condition. There are a number of very cogent considerations, founded upon the expert evidence, which support this theory. In summary they are the following:

(a) The nature of appellant's injury and the treatment received by him were calculated to make him a potential candidate for a Volkmann's of the Holden type 2 variety. As Prof. Du Toit put it, "Volkmann's was a special risk in this case". This is mainly because (i) the fractures and the associated tissue injury were situated in

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the upper forearm, a type of injury which apparently ranks second only to fractures about the elbow as a cause of the development of a Volkmann's; (ii) the fracture of one of the bones, the radius, was a comminuted one; (iii) the trauma of the accident, the manipulative procedures involved in the attempts at a closed reduction of the fractures and the surgical procedures employed in the open reductions would have caused a substantial amount of tissue damage in the vicinity of the fracture sites, with resultant post-operative swelling and a measure of interference with the blood supply to the affected tissue; and (iv) the application of a circumferential (and unsplit) plaster cast could cause a degree of constriction, should the arm swell, and this could contribute to the build-up of pressure in the osteofascial compartments.

/ (b) .....

(b) The ultimate appearance of appellant's arm and hand was indicative of an ischemic contracture. Prof. Solomon stated that the deformity with which appellant ended, making allowance for the fractures and the operations, was quite characteristic of an ischemic contracture of the forearm. Prof. Solomon first saw appellant's arm exactly three months after Dr Boonzaaier's operation on Friday 28 May and then diagnosed an ischemic contracture. He at no time deviated from his diagnosis. His evidence, as the learned trial Judge emphasized, is very important not only for its intrinsic merits but also because of all the medical witnesses, apart from Dr Van Niekerk and respondent, "he was the first on the scene". Dr Cooke was equally positive. He said that the appellant had "the typical, classical Volkmann's ischemic contracture". In this connection he pointed out that one of the

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characteristics of a Volkmann's is that it is selective in its nerve and muscle damage. It tends to cause maximum damage to the median and ulnar nerves, more particularly to the median nerve, with the preservation of the radial nerve. In appellant's case there was some preservation of the radial nerve and total loss of the function of the median and ulnar nerves. Studies by one Seddon had shown that the muscles of the forearm most likely to survive an ischemic contracture were the flexor carpi radialis in the flexor group and the extensor carpi radialis longus in the extensor group. These were the only muscles in appellant's arm in which any function could still be detected. The reason for this selective lesion is that, in a Volkmann's, maximum damage occurs at the centre of the forearm; the deepest structures are destroyed maximally and those at the periphery, near the subcutaneous tissue, tend to

/ suffer.....

suffer the least. Under cross-examination Dr Cooke conceded that appellant's stiff wrist was not part of the classical Volkmann's picture, but pointed out that when joints are immobilised for a long time they become stiff. Also the extent of immobility of the arm was extremely variable in a Volkmann's. He also had to concede that according to Seddon's studies there were other muscles which appeared to have a higher survival rate than those which survived in appellant's case. This latter concession did not, however, in my view, detract from the fact that the two muscles which did survive were peripheral ones and that their survival was suggestive of an ischemic contracture. Dr Cooke's analysis and his view that the ultimate pathology fitted into the picture of a Volkmann's impressed ELOFF J and it impresses me too. I find no real answer to his arguments in the evidence of respondent's experts.

/ (c).....

(c) As has been frequently pointed out, however, the alleged Volkmann's in this case was complicated by sepsis. The theory of appellant's experts was that the ischemia necrosed and devitalised the muscle and nerve tissue and thus rendered it vulnerable to the ravages of the staphylococcus aureus. The fact that there was this underlying ischemic lesion makes plausible and explicable the nature and extent of the staphylococcal infection and the pattern of damage which it left in its train. The theory seems to fit the facts as far as the ultimate condition of the appellant's arm is concerned.

(d) Having regard to the rapidity of the destructive process and the extent of the damage wrought by it, the situation seems to admit of only two possible diagnoses: ischemia with a superimposed sepsis and sepsis alone. To the extent that the improbabilities which I have mentioned weigh down against

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the sepsis theory they tend, see-saw-like, to boost the probabilities in favour of the ischemia-cum-sepsis theory. For there is no other acceptable alternative.

The trial Judge, proceeding on a similar line of reasoning, came to the conclusion that, leaving the clinical history out of account, there were factors and circumstances which established a strong probability that in the week in question appellant had sustained a severe ischemia. After considering the clinical picture, however, ELOFF J came to the conclusion that there were certain features, notably the passive extension test, which tipped the scales the other way and led him to the conclusion that —

> "... it cannot be taken to be more probable than not that plaintiff at the relevant time sustained significant Ischemia."

On appeal, therefore, this is really the crux of the matter. Does the evidence in regard to the clinical history so rebut the strong probabilities favouring the theory advanced by

/ appellant's...

appellant's experts as to non-suit the appellant?

The clinical history and the passive extension test

The evidence as to appellant's clinical history during the week in question presents two basic problems: (i) the conflicting factual evidence as to what symptoms were discernible during this period; and (ii) the conflicting expert testimony as to how this symptom-complex should be evaluated and interpreted.

Two relevant symptoms about the existence of which there can be no dispute are pain and swelling. As I have indicated, the appellant himself deposed to having suffered a lot of pain on Monday and Tuesday. He was in pain when he awoke on Monday morning and the pain got progressively worse during the day. He felt that the tight plaster was causing the pain. As the day progressed his fingers began to swell. In the evening he became more and more uncomfortable. The plaster was feeling tighter on his arm and he was suffering a lot more pain

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than he was in the morning. By Tuesday morning his hand was very swollen. His arm was very painful, more painful than on the Monday. The splitting of the plaster on Tuesday afternoon relieved the pain and tightness on his arm to some extent but he still suffered a pain along the whole arm. This evidence was supported by the testimony of Mrs Blyth senior, who told the Court that appellant was not inclined to complain about pain: "his father had taught him to be a man and not to give in too easily". Nevertheless, appellant told her on Monday morning that his arm was very painful. By lunchtime that day his fingers had become swollen. Appellant kept telling her that he was in great pain. On Tuesday morning his fingers were more swollen. She could see the pain "written on his face".

The bed-sheets covering the period Sunday night to Tuesday evening contain frequent references to pain and swelling. Some of these have already been detailed.

/ Sister.....

Sister Loots, who made a number of these entries, gave evidence. She was called on behalf of the respondent. She initially claimed to have an independent recollection of appellant's case, but under cross-examination conceded that she had no independent recollection of the nursing which he received: she had to rely on inferences drawn from entries which she had made in the bed-sheets. Sister Loots appears to have made a favourable impression on the trial Judge, but a careful reading of her evidence leaves me in doubt as to her objectivity as a witness. For example, she was referred during evidence-in-chief to an entry made early on Monday morning to the effect that an analgesic had been administered "vir erge pyn". In regard thereto she said —

> ".... die pasiënt het gekla van erge pyn, maar dit was nie oormatige pyn nie. Hy het nie vreeslik gekla van pyn nie; hy het net gesê hy het baie seer en kan hy asseblief 'n inspuiting kry."

She was also asked in chief about another entry made by her: "Vingers is baie styf". She stated —

/ "Wanneer....

> "Wanneer n pasiënt se hand geswel is, is daar n mate van styfheid ook teenwoordig, en dit is meer styf in vergelyking met die ander hand, en die ander hand is nie geswel nie en ek het dit geskryf slegs omdat die hand geswel was, was dit moeilik vir hom om te beweeg maar hy het wel beweeg. Dit was nie uitermate styf, hy kon dit nie glad nie beweeg nie. Hy kon sy vingers beweeg".

If, as she later conceded under cross-examination, she had no independent recollection of the nursing received by appellant and was relying upon inferences drawn from the bed-sheets, this evidence is remarkable, to say the least. Her endeavours to water down the entries in the bed-sheets and her other apparent reconstructions smack to me of partiality.

At this point I might just add that I similarly gained the impression that sisters Le Roux and Greyling had taken sides in this case. This was demonstrably so in the case of sister Greyling, whose evidence in Court was far more favourable to respondent's case on several important points than her statement to appellant's attorney.

/ Likewise.....

Likewise sister Le Roux's gratuitous criticism of sister Froneman (to which I have already referred), her evidence that on Monday not only was the plaster fully split but the cotton wool was cut (wholly rejected by me and rejected by the trial Judge as to the cotton wool) and the manner in which she gave that evidence (as detailed above) convince me that she also was not impartial.

There was much debate between the experts as to the quantities of analgesics administered to appellant: whether it was excessive or whether it was normal for fractures of the type sustained by appellant and for the usual aftermath of the operative procedures performed. Another matter in dispute was the degree and nature of the pain associated with an ischemic condition and whether the evidence relating to appellant's pain symptoms was consistent with such a condition. Having studied all this evidence, it seems to me that the quantities of analgesics administered were considerable. Although the intervals between adminis-

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trations varied, it would seem that apart from the pre-operative analgesics, doses of either Pethidine or Doloxene or both were given to appellant on an average of once every four to five hours during the whole of the period of approximately 48 hours commencing from the operation and ending at 8 p.m. on Tuesday. Appellant's experts considered this to be a particularly heavy analgesic dosage. Respondent's experts said it was not. The trial Judge was unable to resolve this dispute. I find myself in a similar position. At the very least, however, the analgesic record satisfies me that during this critical 48-hour period appellant had severe, persistent pain associated with his arm. The record amply confirms the evidence of appellant and Mrs Blyth senior in this regard.

It was contended by respondent's experts, relying on certain authorities, that the pain associated with an ischemic condition had to be "severe, deep, unrelenting

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and poorly localised". This particular description emanated from an article written by Messrs Eaton and Green in a publication entitled "Clinical Orthopaedics and Related Research". Prof. Solomon stated that he knew the authors of this article and that he did not regard them as being "terribly authoritative in this field". In some of the articles cited it was stated that although severe pain was often a symptom of an impending ischemia it was not an invariable symptom. Prof. Solomon himself placed the emphasis on persistent pain not readily attributable to the injury - in this case the fractures and surgical wounds. He said:

> "It is the persistence of pain in a fracture that has been adequately reduced and where you would expect things now to have settled down. If under those circumstances, the patient continues to complain of pain one becomes suspicious. The severity of the pain is not as important because we know that there are vast variations in patients' subjective response."

Dr Cooke, in the same strain, made the point that usually

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it is movement that produces post-operative pain: where the arm is completely immobilised in plaster, then persistent pain suggests a cause other than the fracture itself. Respondent's experts, on the other hand, were inclined to emphasize the severe, deep, unrelenting quality of the pain and expressed the view that there was nothing in the evidence to suggest that appellant suffered this measure of pain.

Having carefully considered all the expert evidence, the evidence of appellant and his mother, the evidence of the bed-sheets and the quantity of analgesics administered, and the evidence that appellant was a healthy, fit, sport-loving young man, inclined by upbringing to stoicism, I am of the opinion that the pain suffered by appellant was probably persistent and severe enough to constitute a positive symptom of a developing ischemic condition.

The trial Judge referred to two factors stressed

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by the defence and concluded that they lent cogent support for the contention that the pain which troubled appellant on Monday and Tuesday was not of the "deep, severe, unrelenting type" which is usually symptomatic of an impending Volkmann's. These were (i) that the analgesic dosages "tapered off considerably" from mid-morning on the Monday; and (ii) that the appellant slept a great deal on the Monday and Tuesday. I do not share the views of ELOFF J in regard to those suggested factors. Apart from the evidence of appellant and his mother as to pain, the bed-sheets indicate that appellant complained of pain or was given an analgesic for pain (which presupposes a complaint) on six occasions between midday Monday and 8 p.m. on Tuesday. In fact at 2 p.m. on Tuesday he was given 2 Doloxene tablets "vir pyn"; and half-an-hour later he again received 2 Doloxene tablets, the entry in the bed-sheet reading: "Kla van erge pyn en ongemak". This hardly seems consistent with a tapering off of pain. It is true that the administration of Pethidine ceased after 11 a.m. on

/ Monday.....

Monday (the last dosage), but, as Prof. Solomon remarked —

> "If somebody has never had Doloxene in his life and you give him two Doloxenes it can knock him right out".

It is also true that the evidence indicates that appellant slept fitfully during Monday and Tuesday, but in this connection account must be taken of the soporific effect of the drugs which were being administered. Prof. Du Toit was of the opinion that the analgesics administered on Sunday night would have left appellant "stuporous" by Monday morning. It was also conceded by respondent's experts, notably Dr Van Wyk and Dr Wienand, that pain is a variable factor. Different people have different subjective responses to a situation calculated to cause pain. This was also the evidence of Dr Cooke and Prof. Solomon. Furthermore, account must also be taken of the evidence that appellant was exhausted at the time of the polo match and would, therefore, have been more susceptible to the soporific effect of the drugs.

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Accordingly I am not persuaded that the analgesic history or the appellant's ability to sleep fitfully are inconsistent with the pain suffered by him having been symptomatic of a developing ischemia.

Another positive symptom - and a cause - of an impending ischemia is swelling of the forearm. Of this there is abundant evidence in the instant case. Appellant's hand and fingers were swollen and he complained that his arm felt constricted in the plaster. Respondent and his witnesses have sought to contend that the swelling was not abnormal. I am unimpressed by this. There are references to swelling in the bed-sheets and it was principally abnormal swelling that caused sister Greyling and, probably, sister Froneman to telephone respondent on Monday morning. The fact that the plaster was split on the Tuesday also tends to confirm that there was abnormal swelling causing discomfort. The trial Judge also found that the swelling was abnormal.

/ Appellant....

Appellant and his mother stated that at a certain stage appellant's fingers became bluish in appearance. This was disputed by respondent and certain of his witnesses and, as I have indicated, the trial Judge found (and this finding was not challenged on appeal) that this had not been proved. I do not understand the learned Judge to have held that the evidence established that the fingers did not turn blue. Consequently, the position in regard to this potential factor is neutral. It cannot be said that the fingers were blue and that this was a positive symptom in favour of an ischemia; nor can it be said that the fingers were not blue and that this was a positive symptom against ischemia.

Another of the classical symptoms, loss of sensation or para-anaesthesia, was only shortly canvassed in the evidence. Appellant stated that on Tuesday his arm and fingers were painful and that he could not recall sensation in his fingers. On the Friday Dr Boonzaaier tested his sensation by pricking his fingers and again

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at the Rand Clinic. Respondent alleged that one of the routine tests which he performed each time he visited appellant (including the occasion when he removed the plaster, i.e. on Thursday morning) was to check for loss of sensation in the hand. The same averment was made by Dr Van Niekerk, who visited appellant on Wednesday morning and Thursday evening. Neither of them noted any untoward sign indicating loss of sensation. Yet when Dr Boonzaaier examined the appellant on Friday morning there was, according to respondent, a loss of sensation on the dorsum of the hand. This may have been a sudden development not detectable the day before; or it may point to respondent and Dr Van Niekerk not having tested for loss of sensation properly or at all. Unfortunately this aspect of the matter was not fully canvassed. On balance I am inclined to think that appellant's evidence constitutes an indication, albeit a faint one, that a condition of para-anaesthesia, consistent with a Volkmann's,

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did develop or begin developing during the critical period.

Finally I come to the passive extension test, which, as I have indicated, finally tipped the scales against appellant in the Court a quo. Together with this must also be considered the evidence that appellant was able to move his fingers actively during the critical period.

The medical experts were in agreement that the passive extension test is a most important clinical aid in the detection of an impending Volkmann's. The theory underlying the test was fully expounded by them. Reduced to its essentials the theory is that when the muscles of, say, the forearm begin to be affected by an ischemia, certain chemical changes cause them (or at any rate those that have not lost their motor function) to go into spasm and to contract. Since the flexor muscles are usually more affected than those of the extensor group, the spasm

/ causes....

causes the fingers of the arm concerned to assume a flexed position. The passive movement of these fingers into an extended position stretches the muscles in spasm and this causes pain or, at any rate, a marked increase in pain in the muscles of the forearm. Thus far there is no disagreement between the experts. Their evidence is not in complete accord, however, as to (i) how the test should be performed, and (ii) how infallible the test is.

In regard to these disputed issues very positive opinions were expressed by the experts. Nevertheless, it is noteworthy that their actual experience of a developing Volkmann's of the forearm and of the passive extension test on the fingers was very limited and that they generally tended to base their opinions on what they had read in the medical literature on the subject. Thus, for example, appellant's experts were generally of the view that to be fully effective the test required the full extension, or straightening, of the fingers; whereas respondent's experts

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expressed the opinion that extension to a position illustrated on a drawing, Exh. "D", was sufficient. Exh. "D" was an illustration of the amount of extension which the appellant demonstrated in evidence as having been actually performed by respondent when he visited him. I did not understand any of the experts to be able to say that from his own personal experience, in a comparable case, extension to what was termed, for convenience, "position D" would be an effective test or vice versa. Their evidence was rather based upon their personal interpretations of a number of somewhat equivocal statements in the literature. One point upon which there did appear to be agreement was this: that the condition of ischemia is a progressive one and consequently extension of the fingers to the same position would, over a period of time, become progressively difficult or more painful. In regard to the infallibility of the test, Prof. Solomon stated that it was not completely reliable, but this opinion appeared to be related to cases

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where there was no ischemic pain as such. Prof. Du Toit, on the other hand, agreed that it was "the really important test of incipient Volkmann's" and conceded that if appellant had "full passive extension of his fingers without pain" on Monday and Tuesday he was not getting a Volkmann's at that stage. Dr Cooke pointed out that it was not an infallible test where there is nerve damage producing loss of sensation to the muscle supply, but this would not appear to apply where the patient, as in appellant's case, actually suffered an ischemic pain. Dr Cooke did, however, make one relevant point. This was that where, as in the instant case, there was apparently an ischemia affecting both the flexor and the extensor groups of muscles, so that both groups would be affected by spasm and contraction, this would tend to bring the patient's fingers into a straighter position than if only the flexor muscles were affected. This would tend to make the test less reliable

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and could result in position D representing only minimal stretch. Not unexpectedly respondent's experts laid great emphasis on the passive extension test.

Associated with this was the ability of appellant to actively move and extend his fingers. Both Prof. Du Toit and Dr Cooke agreed that if on Monday and Tuesday appellant had had the full mobility of his fingers and there was full passive extension of his fingers without pain, a Volkmann's was probably not present; or, if present, could not be diagnosed.

The next question is: was the passive extension test performed and, if so, with what result? It is respondent's case that the passive extension tests were performed by himself and Dr Van Niekerk, with negative results. I do not think that Dr Van Niekerk's evidence in this connection carries any weight. The trial Court did not accept that he visited the appellant on the crucial days, Monday and Tuesday. As regards the respondent,

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there are a number of general reasons for doubting the reliability of his evidence. In short, these are:-

(i) The very unsatisfactory evidence which he gave in regard to the splitting of the plaster, its irreconcilability with certain of the pleadings and the very lame explanations which he gave in this connection. This has already been detailed.

(ii) The evidence he gave in regard to Exh. "B", the sketch made by him shortly after the operation for the benefit of Mrs Blyth senior, and in order to explain to her what had occurred. He admitted having made the sketch but denied having written certain words on this document, including the word "shattered", which appeared to relate to the radius. The trial Court rejected his evidence on this point: rightly so in my view. The denial was clearly linked with his evidence that the fracture of the radius was a simple, not a comminuted one. This evidence was also rejected by

the trial Court. As I have already indicated, in this regard, too, I am in complete agreement with ELOFF J.

(iii) In the course of his evidence respondent stated that, although he was present at the operation performed by Dr Boonzaaier on the Friday, he could not recall having put any questions to Dr Boonzaaier and did not discuss the case with him, either during the course of the operation or immediately thereafter. I find it very difficult to accept this evidence. Appellant was respondent's patient; it was obvious from what transpired at the operation that something catastrophic had happened; and I cannot imagine that respondent could have been so indifferent to his patient's welfare as not to discuss the case fully with the specialist who had performed the operation. It is perhaps idle to speculate about the reason for

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respondent's lack of candour on this point. Perhaps he wished to avoid being cross-examined on Dr Boonzaaier's opinion on the case; perhaps his object was to evade the question, as far as possible, as to why at that stage he did not tell the Blyth family how serious the situation was. At all events, this evidence also reflects adversely on his credibility.

(iv) Respondent's evidence in regard to what have been referred to in this judgment as "bed-sheets" was most unconvincing. The bed-sheets were referred to frequently in evidence. In the evidence of the nursing sisters, who were called before respondent and gave their evidence in Afrikaans, the term used was "bedkaart". According to sister Le Roux the patient's bed-sheet was kept in the nurses' duty room ("dienskamer") and according to sister Greyling the temperature chart was also kept there. According to Dr Van Niekerk the procedure

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on ward rounds would include looking at the bed-sheet to see whether there was a temperature present and what medication had been prescribed. Respondent himself referred in his evidence-in-chief and in cross-examination to the patient's "bedkaart" and in the context there is no doubt that he was indicating the bed-sheet. In the course of cross-examination he was asked:

> "Het u ooit na die susters se..... verpleegsters se dienskamer gestap by enige besoek wat u afgebring het om na die bedkaart te kyk?-- Die bedkaart hang daar, so ek het hom elke dag daar as ek gaan het ek hom gesien.
>
> So, met ander woorde, u het self van die bedkaart verneem wat... (tussenbei) -- Presies aan die gang is.
>
> Van tyd tot tyd plaasvind?-- Ja.
>
> U was nie tevrede om net die versekering deur die verpleegster te aanvaar nie?-- Nee."

At a later stage he was cross-examined about a very suggestive entry in the bed-sheet by sister Heyns at 6 a.m. on Wednesday morning. He denied having read this when

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he visited appellant on Wednesday evening. He then stated that this bed-sheet was not a "bedkaart" and that what he read each time he visited the patient was the temperature chart:

> "Nou wat het u deurgelees met elke besoek aan die pasiënt?— Ek het gekyk na die bedkaart, die kaart.... die temperatuurkaart wat voor op hierdie, die bedkaart is.
>
> O, deur bedkaart bedoel u die temperatuurkaart?— Die temperatuurkaart.
>
> U het nooit na hierdie geskiedenis gekyk nie?— Nee, dié dra die suster oor aan enige.... enige verandering hier dra sy oor aan my."

The inconsistency of this evidence is manifest.

Apart from general criticisms of respondent's testimony there are also particular reasons for questioning the reliability of his evidence relating to the performance by him of the passive extension tests. That he did handle and passively move the appellant's fingers is common cause, but I have grave doubts as to whether in doing so he was consciously performing a passive extension

/ test.....

test. He was challenged on this in cross-examination:

> "Die betoog van die Eiser gaan wees, dokter, dat op gn stadium gedurende die Sondag, die Maandag, die Dinsdag en die daaropvolgende dae was u aandag ooit op die moontlikheid van n iskemiese toestand toegespits?-- Dit is sy bewering, ja.
>
> Dit sal ook betoog word dat u nooit, nòg u nòg u vennoot, ooit die toetse en in besonder die passiewe ekstensietoets toegepas het nie?-- Nee, ons het dit toegepas.
>
> En die moontlikheid van n dreigende of n ontwikkelde iskemie was nooit in u gedagte gewees nie?-- Edelagbare, soos ek u weer sê, dit was nie my... die eerste gedagte in my kop nie, maar ek het wel besef dat so n ding kan ontstaan."

This amounts to a concession that at least the danger of ischemia was not in the forefront of his mind. His claim in evidence that he could recall the tests performed by him on each visit to appellant contrasts strangely with the floundering evidence given in regard to the splitting of the plaster. And in regard to all these matters it must be remembered that respondent in evidence was endeavouring to recall the details of routine visits to a

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particular patient (one of many) which had taken place about six years before; that there were no contemporary records of the tests performed; and that it was only about three years after these events that summons was issued and respondent made to realise the importance of these details.

Another factor which would seem to point to the ineffectiveness of any tests performed by respondent arises from his visits on Wednesday and Thursday. It was put very explicitly in the cross-examination of Prof. Du Toit that respondent's evidence would be that when he performed the passive extension test to the fully extended position on Monday, Tuesday, Wednesday and Thursday, there was no increase in pain. This too appears to be the evidence which respondent gave, although he tended to speak generally of the "toetse" performed by him. Now, it is not disputed that appellant had a massive sepsis, which raged during Wednesday and Thursday. It was the opinion

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of Prof. Du Toit that if there were sepsis appellant —

> "... would have had a great deal of pain and in fact the slightest movements of his fingers, especially of extension, would have given agonising pain if he had sepsis.... That is a characteristic of sepsis."

This does not seem to have been really disputed by the other experts. In fact Dr Leitch (called by respondent) made this concession:

> "If the passive extension test had been applied to the Plaintiff on the Wednesday and the Thursday, would you have expected it to elicit marked pain from the Plaintiff?-- Not marked pain. I think he would have had more pain than had he not been infected."

If, as respondent suggests, his examination of appellant's fingers on Wednesday and Thursday was unaccompanied by any increase in pain, then the probabilities would seem to point to the passive extension test not having been performed properly or at all. It is clear from the evidence that appellant was on those days still experiencing

/ considerable....

considerable pain in his arm so that there is no room for the contention that by then his sensory perceptions had become so blunted as a result of nerve damage, as to make him incapable of feeling an increase in pain.

Another point, admittedly of minor importance, is the fact that during the critical period of Monday and Tuesday there was a 24-hour gap in respondent's visits to appellant. If there was an ischemic condition, it was one of rapid development and it seems doubtful whether examinations so widely spaced would have enabled the respondent to gauge effectively appellant's condition and to make the necessary comparisons.

It was argued on behalf of respondent that, even if the latter's evidence be ignored, the appellant's own evidence showed that there had been an effective application of the passive extension test without an increase in pain. It is true that appellant deposed to his fingers having been passively extended by respondent to position

"D". He did not state whether this caused an increase in pain and respondent's counsel refrained from asking whether this caused pain. There was some debate as to the contention that, since respondent wished to rely upon the passive extension test as a decisive clinical finding, it was incumbent upon respondent's counsel to establish from the only person able to depose directly thereto, viz. appellant, that the test elicited no marked increase in pain. I think that this contention has some merit, but I do not find it necessary to decide the point. I have considered the arguments based upon the evidence given by appellant on the assumption that there was no marked increase in pain and my conclusion is the following.

This evidence does raise a probability in favour of respondent. In evaluating the cogency of the probability, however, several factors must be taken into account. These are:

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(1) Position "D" upon which so much debate centred was merely a graphic illustration of a finger position which appellant endeavoured to recall six years after the event. At the time when these "tests" were performed the appellant was in great pain, heavily sedated and, on his own evidence, at times not fully conscious of what was happening around him. In the circumstances, to attach critical significance to position "D" would, in my opinion, be unrealistic.

(2) The point made by Dr Cooke, to which I have already referred, that in this case, because of the affection of the extensor group of muscles, the natural position of appellant's fingers may well have been straighter than otherwise and position "D" could thus have represented only minimal stretch.

(3) The general uncertainty of the expert evidence

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in regard to what precisely the effect of extension to position "D" would have been, had there been an ischemic condition; and the difficulty, therefore, of according such a test a decisive role.

(4) The probability raised by this evidence is just one of many which must be evaluated and weighed together in deciding the ultimate question as to whether appellant suffered a Volkmann's or not. I shall return to this point shortly.

The other symptom upon which respondent relied was the alleged ability of appellant to actively move his fingers during the period in question. I do not think that the evidence establishes an ability to extend his fingers fully, the test referred to by Prof. Du Toit and Dr Cooke as negativing an ischemic condition. Appellant and his mother certainly did not depose to such

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an ability: quite the contrary. Appellant could not recall being able to move his fingers on Monday morning, although they may have moved fractionally. He tried to move his fingers on Tuesday morning and cannot recall having seen them move. The position was the same on Wednesday. According to Mrs Blyth senior, there was "the tiniest little flicker" when appellant was asked to move his fingers on Monday morning. He never moved his fingers after that. If there was any movement, it was the "tiniest of flickers". Respondent's evidence on the topic is somewhat vague. In chief he stated that he thought that on Sunday night, after the operation and after appellant had regained consciousness, he asked appellant to move his fingers and that as far as he could remember appellant did move his fingers. In answer to a general question as to the tests employed by him to satisfy himself in regard to the patient's circulation, he referred to a number of tests, but not the active movement of the

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fingers. He was then referred to the movement of the fingers and asked —

> "Speel dit enige rol? In die dophou van die pasiënt?-- Ja, ja. 'n Mens... hy moet sy vingers flekseer en ekstendeer.
> Die passiewe ekstensie en fleksie van 'n pasiënt se vingers?-- Ja?
> Wat verstaan u daaronder? Speel dit enige rol in die dophou van 'n pasiënt?-- Ja, dit speel 'n groot rol by verdagte iskemie."

(Here active mobility and passive extension and flexion appear to be confused.) Later he stated that on Monday morning appellant "het self sy vingers beweeg... daar was definitief goeie motor funksie". No specific detail is given, however; and this evidence stands in contrast to the notation on the bed-sheet, made about 5½ hours earlier - "Vingers is baie styf". On Tuesday he again asked appellant to move his fingers, as one of a series of tests and found everything normal. On Wednesday night he again did the usual tests and found appellant's circulation to be good. On Thursday morning he performed the same routine

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examination: the circulation was normal.

My general comments in regard to the reliability of respondent's evidence as to the execution of the passive extension test apply with equal force to this evidence relating to the active movement of appellant's fingers. In addition this latter evidence is vague. I also find it difficult to accept that on Thursday morning, when the sepsis was raging and by when a good deal of damage to appellant's forearm must have been wrought, a properly executed test as to finger mobility would have shown the position to be normal.

It is true that there are a number of references in the bed-sheets to appellant moving his fingers or exercising his fingers, to being encouraged to move his fingers and to his circulation being satisfactory. It is to be noted, nevertheless, that these kinds of observations continue through the week. Thus, for example, even on Thursday

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night there is an entry: "Vingeroefening is gedoen"; and on Friday night: "Vingers en hele hand is 2-uurliks beweeg". But in no instance is precise detail given and, for the reasons already stated, I do not think that the evidence of the nurses themselves takes the matter any further. In fact, the only time anything precise is recorded is on Friday when Dr Boonzaaier evidently issued instructions that "albei vingers van hand moet geoefen word deur vingers reguit te strek". To my mind the evidence of active finger movement is too vague and unsatisfactory to be of real diagnostic value.

In the final analysis, therefore, the issue as to whether appellant suffered a serious and generalised ischemic condition in his forearm must be determined by putting in the balance, on the one hand, the probabilities favouring such a diagnosis to which I have already referred and, on the other hand, the probability against such a diagnosis flowing from the evidence concerning the passive ex-

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tension of appellant's fingers. Having carefully weighed the position, I am satisfied, for the various reasons already stated, that there is a substantial preponderance in favour of appellant's case and I hold that it is more probable than not that appellant suffered a severe and generalised ischemia in his right forearm; that this ischemia so devitalised the muscle tissues of the forearm that it was possible for the staphylococcal infection to become a massive and invasive one; and that as a result thereof there was a large-scale destruction of muscle and nerve tissue and ultimately a fairly typical Volkmann's contracture. This finding of course reverses that of the Court a quo. The next question is whether this eventual result can be attributed to negligence on respondent's part.

NEGLIGENCE

The trial Judge found that the respondent had acted negligently in a number of respects. These were:-

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(a) That the plaster cast or the underlying padding was not suitable and too tight and should have been split, probably shortly after the operation. On all the evidence the defendant should have realised that the omission to do so might contribute to the build-up of pressure in the forearm and induce ischemia. In failing to realise this respondent was negligent.

(b) That once respondent was told by the nursing sisters on Monday that the swelling was out of the ordinary, he should have gone to see the appellant as soon as possible in order to satisfy himself that a compartmental pressure was not creating an ischemic condition, to ensure that the splitting of the plaster and padding was effectively done, to examine the forearm and to do all the necessary tests to make certain that there was no vascular embarrassment. In failing to do this

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he was negligent. In this connection the trial Judge preferred the evidence of appellant's experts to that of respondent's experts.

(c) Possibly, that he paid insufficient attention to the swelling of the plaintiff's fingers.

Despite these findings, it followed from the trial Judge's conclusion to the effect that a general ischemia did not take place that his negligence was not causally connected with the ultimate catastrophe and consequently did not found a cause of action based on damage suffered as a result thereof. In view of this Court's finding that there was such a general ischemic condition the causal obstacle which confronted the trial Court no longer applies. It must nevertheless be determined whether causal negligence was established. And, as I have pointed out, by reason of concessions made by appellant's counsel the enquiry is limited to respondent's post-operative care of appellant. It is also clear from the submissions of appellant's counsel

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that the general complaint against respondent is that he failed to take action which in the circumstances he could reasonably have been expected to take, i.e. the case is based on negligent omissions.

Applying the basic principles relating to delictual negligence which is causally linked to the damage suffered to the situation in the present case, it seems to me that this enquiry resolves itself into the following questions:

(i) Whether the reasonably skilled and careful medical practitioner in the position of the respondent would have realised that a serious ischemic condition was developing or threatening to develop in appellant's forearm; and, if so, when he would reasonably have come to realise this.

(ii) Whether there was remedial action which could reasonably have been taken.

(iii) Whether the same notional practitioner would

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have known of this remedial action and would have realised that it had to be taken.

(iv) Whether the remedial action, if taken when the need for it ought reasonably to have been realised would have prevented the damage suffered by appellant.

(v) Whether respondent himself failed to take such remedial action.

These questions raise a number of issues upon which the expert medical evidence was again sharply divided. It will, therefore, be necessary for this Court to attempt to resolve these differences of opinion and where possible to make findings on the issues, based upon the probabilities. In this regard some assistance will be derived from the conclusions of the Court a quo.

As to question (i), it is clear to me that the reasonably skilled and careful medical practitioner in the

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position of respondent would have been aware of the danger of an ischemic condition developing in appellant's forearm. He would have known that this danger was a dual one, i.e. it could arise by reason of arterial occlusion or embarrassment or because of the development of a compartmental syndrome. In view of the various factors previously mentioned - the site and nature of appellant's fractures, the degree of trauma involved as a result of not only the accident itself but also the manipulative and surgical procedures applied, and the application of a circumferential (unsplit) plaster cast - the reasonably skilled and careful practitioner would have realised that the development of a compartmental syndrome was a special risk in this case. This would have placed upon him a duty to be especially careful to watch for any untoward signs that might point to the development of an ischemic condition and to act immediately if any such signs became apparent.

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The role of a circumferential and unsplit plaster cast in the build-up of a compartmental syndrome was one topic upon which the experts were not wholly ad idem. Their differences were, however, not ones of fundamental principle, but rather of emphasis. Appellant's experts tended to attribute to such a cast a rather more important contributory role than did respondent's experts. It is a matter of degree. There seems little doubt that in a severe compartmental compression, characterised by rapid swelling and pressure build-up, a too-tight circumferential plaster may well contribute significantly to the total syndrome. This is something which the reasonably skilled and careful practitioner would appreciate.

The compressive effect of a plaster cast may be neutralised by splitting and spreading or, even better still, by bivalving the plaster and keeping it in position by means of a crèpe bandage. The disadvantage of interfering with a plaster in this way is that it disturbs,

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or may disturb, the reduction and immobility of the fractured bones. Where, however, as in this case, the one bone has been plated this danger is diminished substantially. And, in any event, the possibility of a Volkmann's, with all its disastrous effects, generally outweighs the disadvantages of a splitting. There is a school of thought which believes that whenever an operation is performed on a forearm and the arm is encased in a circumferential plaster cast, the cast should be completely split immediately, i.e. before the patient returns to the ward, in order to obviate vascular embarrassment and the build-up of compartmental pressure. Dr Cooke in particular subscribed to this school of thought and there certainly appeared to be considerable support for this approach in the medical literature. Prof. Solomon considered it to be "good practice", both at the time when he gave his evidence (i.e. in 1977) and in 1971. Dr Cooke even stated that the application of a circumferential plaster

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to such a fresh fracture is negligent if the plaster is not then split. This was strenuously disputed by respondent's experts. I do not find it necessary to resolve this particular issue. In my view, the immediate splitting of such a plaster is a safe and prudent practice, but I would hesitate to say that a practitioner who fails to do so is necessarily negligent. But if he does not do so, then I think that the situation casts upon him a duty of especial vigilance. He must then watch the patient, or ensure that the patient is watched, very carefully for signs of a threatened ischemia. At the first suspicion of ischemic complication he must act immediately and the first step would be the splitting and, possibly, the removal of the plaster. I shall have more to say about this when I deal with question (ii) above. I might just add that in so far as the above-stated conclusions run counter to the expert evidence adduced by respondent, I have preferred the views of appellant's experts, parti-

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cularly those of Prof. Solomon.

Reverting to the essence of question (i) I am of the opinion that a reasonably skilled and careful medical practitioner in the position of appellant would have realised on the Monday that there were symptoms which, even if they did not demonstrate a developing ischemia, raised a real suspicion that one might be developing. For the reasons already stated he would have been particularly vigilant for such symptoms and even at 8 a.m. when he first saw appellant he would have been alerted, firstly by the persistent pain and discomfort being suffered by appellant and, secondly, by the notations in the bed-sheets. In regard to the bed-sheets I have in mind particularly the observation at 2.30 a.m. that appellant's fingers were "baie styf" and the entries in regard to pain and the analgesics administered. These suspicions would

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have been strenghtened, if not confirmed, when later on Monday morning he was telephoned by two nursing sisters and told that the appellant's arm and hand were abnormally swollen. The situation called for immediate action. In reaching these conclusions I have again rejected what appear to me to be the somewhat conservative views of respondent's experts.

Turning to questions (ii) and (iii), the remedial action which the notional practitioner could and should have taken in the circumstances was described by Prof. Du Toit, Dr Cooke and Prof. Solomon. In essence it amounted to this. The very first step would be to remove the plaster or at any rate split it completely and expose the skin. This in itself has two advantages. If the plaster is constricting the arm, removal or splitting will bring relief. Secondly removal or splitting enables the practitioner to examine the arm and to see

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what is occurring underneath all the dressings. The doctor is then able to see whether the arm itself appears swollen or whether from the appearance of the skin there seems to be swelling and compression within the fascial compartments. Furthermore, the usual tests for ischemia, designed to detect the five P's and the passive extension test, would be performed. Thereafter the patient's condition would be carefully watched and if the adverse symptoms persisted, then the more drastic step of a fasciotomy would have to be considered, and, if necessary, performed.

Turning to question (iv) whether the remedial action, if taken when the need for it ought to have been realised, would have prevented the damage suffered by appellant. This point was not as fully dealt with in the medical evidence as certain other aspects. It was conceded by appellant, by way of a formal admission made by counsel during the course of the trial, that the ischemic condition in appellant's forearm became irreversible by

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6 p.m. on Tuesday 25 May - this too was the general effect of the evidence of appellant's medical experts - and that the negligence alleged against respondent was limited to the period between 6 p.m. on the Sunday and 6 p.m. on the Tuesday. Looking back in retrospect and with limited contemporary records, it is difficult for any expert witness to say precisely when the condition became irreversible or when the proper ~~xx~~ remedial action, if taken, would have prevented or avoided the injury to his forearm which the appellant ultimately suffered. Viewing the evidence as a whole, however, I think it establishes as a matter of probability that had respondent been alerted by his own observations to the danger of an impending ischemia on Monday morning, either when he saw appellant at 8 a.m. or later in the morning when he was telephoned, and taken the appropriate remedial action, as detailed above, then the severe and generalised ischemic condition with concomitant tissue necrosis would have been avoided. This would have

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prevented the staphylococcus aureus infection from spreading in the way in which it did: it would have been sealed off and localised by the body's natural defensive responses. The likelihood, therefore, is that there would have been no large-scale muscle destruction and certainly no nerve lesion. The appellant might have had two unpleasant abscesses in the region of the surgical wounds, but there it would have ended. More probably than not the fractures would have healed satisfactorily and appellant would have regained the full use of his arm.

As to question (v), it is clear that the requisite remedial action was not taken by respondent. This is partly because he did not diagnose an impending ischemia or suspect the possibility of one developing. In failing to do so, he was, therefore, negligent in that he failed to display the skill and care reasonably to be expected of him. Another reason why he failed to make the appropriate diagnosis was because he did not maintain the necessary vigilance (he

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allowed 24 hours to elapse between visits during this vital period) and when he was telephoned by the sisters and told of their concern he did not go to see things for himself. That was the stage *par excellence* when he should have hurried to the hospital, removed the plaster and commenced the remedial procedures detailed above. In failing to do this and in particular in leaving the splitting of the plaster to the nursing staff he failed in his duty towards his patient and was negligent. In the result the plaster was not properly split on the Monday and even on the Tuesday when the plaster itself was split, the padding was not cut. In essence, therefore, I am in agreement with the findings of negligence come to by the Court *a quo*. In addition (and contrary to the findings of the Court *a quo*), for the reasons aforestated, I am of the opinion that the negligence was causally connected with the damage ultimately sustained by the appellant.

That conclusion establishes the liability of the

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respondent to appellant. The only remaining matter is the question of the quantum of damages.

THE DAMAGES

The trial Court made no assessment of the damages. In view of its conclusion it was not necessary for it to do so. Counsel were agreed that, if we should decide to uphold the appeal and allow appellant's claim, we should ourselves compute the damages claimable.

Appellant's claim for damages was advanced under a number of heads. These heads and the amounts claimed at the appeal stage were the following:-

(1)	Medical expenses to date	R673,70
(2)	Estimated future hospital and medical expenses	2 868,60
(3)	Costs of an electronic arm	9 497,00
(4)	Reduction in earning capacity	52 630,00
(5)	Pain and suffering	10 000,00
(6)	Permanent disability, disfigurement and loss of amenities	30 000,00
		R105 669,30

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The first two heads represent items upon which the parties reached agreement. The first is self-explanatory. The second relates to the estimated cost of certain reconstructive surgery designed to restore maximum function to appellant's arm without amputating it. Respondent conceded that this was a worthwhile procedure and agreed to the amount claimed.

Head (3) relates to the cost of an electronic arm, should appellant ultimately agree to an amputation of the arm. Appellant's counsel submitted that appellant would probably undergo an amputation in the future and that a reasonable approach would be to anticipate that an amputation will either become necessary or acceptable by the time appellant reaches the age of 31 (he is now 28). Upon the basis that the expenditure on the electronic arm (or prosthesis) would commence in three years' time, the amount required for the electronic arm and replacement arms up to the age of 70, capitalized at an agreed rate of 3% per annum, was calculated

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as being the sum of R9 497,00. Respondent's counsel argued that it was "improbable in the extreme" that appellant would ever undergo an amputation and that no allowance should be made for either the cost of the amputation or the electronic arm. I did not understand respondent's counsel to challenge the figure of R9 497,00, should appellant accept an electronic arm at the age of 31.

It is true that hitherto the appellant has set his face against an amputation. The possibility of appellant's arm having to be amputated was first mooted by Dr Boonzaaier on 4 August 1971. Thereafter the question was raised on a number of occasions by the various medical men consulted by appellant. Appellant nevertheless decided to retain his arm. In their joint report appellant's medical experts (Prof. Du Toit, Dr Cooke and Dr Sacks) expressed the view that amputation and the fitting of an electronic arm was the correct treatment for his right arm condition. When they came to give evidence they all qualified their

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viewpoint and opted in favour of the proposed reconstructive surgery, leaving amputation as a residual possibility. Appellant's attitude at the time of the trial was that at that stage he preferred to retain his limb but that, if a situation developed necessitating an amputation, he would agree to that. Prof. Du Toit pointed out that there were various causes which might necessitate an amputation, e.g. sepsis developing in the course of the proposed reconstructive operations, a severe trophic ulcer from a burn or other injury and other similar complications. He assessed the risk of this at 10% or 15%. In addition there is the possibility that appellant may change his mind and no doubt scientific developments in the future may make the alternative of an artificial arm more attractive. I infer from appellant's evidence that one of the factors weighing with him at the time of the trial was that he had endured very considerable pain and suffering in connection with his arm and did not wish, voluntarily, to undergo more pain.

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According to Prof. Du Toit the amputation would cause severe pain for the first day or two and then a rapid diminution of pain. As time passes and the memory of past suffering fades appellant may well view more favourably the amputation alternative.

To sum up, I think that there is a distinct possibility that appellant may at some stage in the future either be compelled to have an amputation and an artificial arm or voluntarily opt in favour of this form of treatment. The situation is, however, full of imponderables. Apart from the chances of this ever happening, there is the imponderable as to when it will happen — and this, of course, will have a direct bearing upon the number of electronic arms which he will require and the cost thereof.

The practice of our courts in assessing damages in a situation such as this was well stated by COLMAN J in Burger v Union National South British Insurance Co., 1975 (4) SA 72 (W), at p 75 D - G, as follows:

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"A related aspect of the technique of assessing damages is this one; it is recognised as proper in an appropriate case, to have regard to relevant events which may occur, or relevant conditions which may arise in the future. Even when it cannot be said on a preponderance of probability that they will occur or arise, justice may require that what is called a contingency allowance be made for a possibility of that kind. If, for example, there is acceptable evidence that there is a 30 per cent chance that an injury to a leg will lead to amputation, that possibility is not ignored because 30 per cent is less than 50 per cent and there is therefore no proved preponderance of probability that there will be an amputation. The contingency is allowed for by including in the damages a figure representing a percentage of that which would have been included if amputation had been a certainty. That is not a very satisfactory way of dealing with such difficulties, but no better way exists under our procedure.

I would refer, in regard to this aspect of the matter, to the remarks of WESSELS, J.A., in Van Oudtshoorn v Northern Assurance Co. Ltd., 1963 (2) S.A. 642 (A.D.) at pp. 650-651."

In this connection COLMAN J drew a distinction between causation and quantification and observed that it had never been the approach of the court, when faced with uncertainties in

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regard to the consequences of injury and the quantification of the loss suffered, to resolve these uncertainties by the application of the burden of proof. Although, as COLMAN J conceded, it is not always possible to distinguish clearly between causation and quantification in this sphere, I agree that this distinction underlies and justifies the general practice of taking into account certain future possibilities, which have not been shown to be probabilities, in computing prospective damages. (See also Kwele v Rondalia Assurance Corporation of SA Ltd., 1976 (4) SA 149 (W), at p 152 H to 153 A.)

In the present case I do not propose to express the possibility of appellant undergoing an amputation and having an electronic arm fitted at some time in the future in terms of a precise percentage. I intend merely to award an amount in respect of the total cost of the amputation and electronic arm which will take account of the extent of the possibility and the various imponderables to which I have referred. Appellant's heads do not refer to the cost

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of the amputation, but from the evidence it appears that at present the operation would cost about R350, to which must be added hospitalization for anything between 5 to 14 days, say 10 days, at a cost of R22,50 per day, viz. R225, making a total of R575. The capitalized cost of the electronic arm, as stated above, is R9 497. In all the circumstances, I think that it would be fair to award R3 500 under this head.

The claim in respect of reduction in earning capacity (head (4)) was computed in appellant's heads of argument and in the evidence on the following basis. Appellant is a farmer. Whether he continues with his present disablement (with the possibility of slight improvement as a result of reconstructive surgery) or opts for amputation and an electronic arm, he will be severely handicapped in his day-to-day farming activities. In order to compensate for this he could be provided with a semi-skilled Black assistant to supplement this deficiency in his working

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effectiveness. The claim of R52 630 represents the capitalized cost of providing such an assistant for appellant until he reaches the age of 70 years. I do not think this is a realistic basis for computing this head of damage. In the ordinary course of his farming activities, appellant would probably have the necessary assistance from his farm labourers in any event. In argument appellant's counsel conceded this and contended that the Court should award an arbitrary amount to compensate appellant for his working disability and his disadvantage in the labour market should he, owing to unforeseen circumstances, be forced to give up farming on his own account. Appellant's counsel suggested an amount of R15 000. Respondent's counsel, on the same basis, submitted that R5 000 would be adequate compensation. In my view, an award of R7 500 would provide fair and adequate compensation under this head.

Finally, I come to heads (5) and (6), covering general damages for pain and suffering, permanent disability,

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disfigurement and loss of amenities. It is convenient to consider all these aspects of appellant's loss together and to make one composite award. Here on the one hand appellant claimed R40 000 and respondent suggested that R 10 000 would provide adequate compensation. There is no doubt that appellant is entitled to a substantial award of damages under these heads. The pain and suffering attributable to the ischemia, the invasive sepsis, the virtual destruction of his forearm and the various remedial procedures which were attempted must have been very considerable. Having a suppurating, septic arm for about 4 months must itself have been a very unpleasant experience. The disability, which is the virtual loss of function of his right arm, is a most serious one. Appellant has nevertheless faced his misfortune with fortitude and has shown a willing ingenuity in adapting to his handicap. Although right-handed, he has learnt to write with his left hand and also

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to play tennis left-handed, using the contracted right hand in some ingenious way to throw up the ball when serving. Nevertheless, there remains a disablement which adversely affects the sports and pastimes such as polo, golf, swimming, dancing, rowing, fishing and weight-lifting of which he was fond. He is handicapped, too, in his daily activities, e.g. dressing himself, bathing himself, cutting his food at table, playing with his young children and so on. Although this Court has not actually seen his arm in its present condition (or a photograph thereof), from the descriptions given it is not difficult to visualize its appearance. It represents a very considerable disfigurement and appellant confessed that he was very self-conscious about it. No doubt, in the course of time, this feeling of self-consciousness will diminish, but it will probably never disappear entirely.

Taking all this into account and bearing in mind that appellant's deprivations came at the early age of 20,

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when he was at the threshold of manhood, I am of the opinion that an award of R20 000 would be appropriate under heads (5) and (6). In arriving at this figure I have also had regard to comparable awards of general damages in other cases and the steady diminution in the purchasing-power of money. In the result, therefore, the damages to be awarded under the various heads are as follows:

(1)	Medical expenses to date	673,70
(2)	Estimated future hospital and medical expenses	2 868,60
(3)	Costs of electronic arm (and amputation)	3 500,00
(4)	Reduction in earning capacity	7 500,00
(5) & (6)	General damages for pain and suffering, disability, disfigurement and loss of amenities	20 000,00
		R34 542,30

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COURT'S ORDER

Having regard to the aforegoing the following order is made:

(1) The appeal is allowed with costs.

(2) The order of the Court a quo is altered to read:

> "Judgment for plaintiff in the sum of R34 542,30 and costs of suit, such costs to include the qualifying expenses of Prof. Du Toït, Dr Cooke, Prof. Solomon, Dr Sacks and Dr Spiro. Plaintiff is declared a necessary witness."

(3) Both in this Court and in the Court a quo the costs of two counsel are allowed.

M.M. CORBETT

RUMPFF CJ)
JOUBERT JA)
GALGUT AJA) CONCUR.
BOTHA AJA)